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PROGRESSIVE DEAFNESS, OTOSCLEROSIS, AND CLOSELY RELATED SUBJECTS. AN ABSTRACT OF THE AVAILABLE LITERATURE PUBLISHED DURING THE YEAR 1938.

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Introduction: Although the term progressive deafness was adopted in order to limit the field of discussion and avoid the consideration of distinctly nerve deafness, in the almost 100 contributions reviewed with the most valuable assistance of my enthusiastic associate, Dr. Louis E. Silcox, constant overlapping in the field of neuro-otology has made the process a difficult one; and in order to observe continuity two or three papers from the 1937 journals and one from January, 1939, have been included.

The chief obstacle, however, lay in the confusion of definition as to progressive deafness and otosclerosis — discussion of these terms with some brilliant otologists brought out the opinion that there was no such entity as progressive deafness — all deafness was essentially progressive in certain periods, and that the term otosclerosis now popularly used was also a misnomer. Otosclerosis, as so accurately portrayed by Cahill, and of so definite a pathologic pattern as observed by Crowe and his associates in their postmortem research, might appear in only a few cases in a thousand, whereas in other more distant clinics throughout the country 70 per cent of the progressive deafness cases with negative Rinné and prolonged bone conduction would be termed otosclerotic. Anyhow, the adjective seems a little less formidable than the noun and it would seem to have been rather uniformly applied to the type of ear considered most favorable for the fistulization procedure. As a

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result, the double title has been selected for this review and no further explanation will be presented.

The articles abstracted seem to fall into three rather classic groups, and this arrangement should simplify the reader's study.

Groups for Consideration: Group 1: Clinical Studies and Research. *Group 2:* Medical Therapeusis and Systemic Readjustment. *Group 3:* Surgical Procedures.

Group 1, Clinical Studies and Research: Nager and Fraser[1] conjointly, while conceding the general knowledge that main changes in otosclerosis concern the labyrinthine capsule with only minor internal ear involvement, in examination of large numbers of otosclerotic petrous bones, in some rare cases found more or less extensive bone formation in the scala tympani. They reviewed several cases, two of them cretins. They found the scala tympani filled up with lamellar as well as otosclerotic bone, atrophy of the organ of Corti, and stria vascularis, swollen and hypertrophic as in acute inflammations of the labyrinth. They did not accept the customary belief that former labyrinthine infection caused the otosclerotic infection but believed the otosclerotic process in labyrinthine wall irritated the endosteal layer and perilymphatic spaces, causing fibrous and circumscribed bone production in the scala tympani. Bone formation was occasionally found in the semicircular canals. They considered it noteworthy that bone formation in the inner ear was only found in otosclerotic diseases of the labyrinthine capsule and not following other infections.

Nager[2] subsequently reported alone in a Swiss journal practically the same material, omitting the special case report. As this practically duplicates the previous article, no further abstract will be given.

Guggenheim,[3] discussing the diagnosis and treatment of otosclerosis, stated that dystrophic bone constituting the otosclerotic focus was laid down during the fetal period and later subjected to the same stimuli from thyroid and pituitary hormones that all bone receives.

He did not consider diagnosis difficult after involvement of hearing mechanism had occurred. Family history was important only if positive — otherwise of no particular significance. He cited the fact that Nager found silent otosclerosis in 5 per

cent of individuals succumbing to accidents. He gave these 12 important signs:

1. Decrease or absence of cerumen.

2. Bilateral condition.

3. Defective or absent vasomotor response of drum membrane.

4. Normal appearance of drum membrane and mobility of membrane and malleus.

5. Patent Eustachian tubes.

6. Negative Rinné test.

7. Prolonged Schwabach test.

8. Paracusis Willisii.

9. Varying degree of tinnitus and throbbing.

10. Slight pain in small percentage of cases.

11. Pink shimmer in a few cases.

12. Normal bone conduction with varying degrees of conduction impairment.

Otosclerotics should avoid propagation because:

1. Offspring may inherit it.

2. Mother may deteriorate in hearing during pregnancy.

The writer discussed canal fistulae of Barany in treatment and unsuccessful removal of footplate in Politzer's day. He then discussed embryologic development of otic capsule and the utilization of the condition around the otic capsule in otosclerosis, called halisteresis, for the so-called therapeutic experiment of reversing the condition around the focus, thus inhibiting the expansion of the dystrophic area. This consists of:

1. Analysis of diet.

2. Correction of diet to age, type of work, etc.

3. Administration of vitamin C — 1,000 to 3,000 units daily in form of orange, lemon and tomato juice, sometimes cevitamic acid.

4. Calcium, 3 to 6 gm. Phosphorus, 6-12 gm. daily, in form of calcium phosphate.

5. Vitamin D — 3,000 to 6,000 units daily, in fish oil fortified with vitamin D.

6. Where tinnitus is present, 1,000-2,000 Sherman units of vitamin B in brewer's yeast, vitamin B complex, or synthetic vitamin B.

Wilson[4] presented a comprehensive article upon the present status of the problem of otosclerosis, first discussing hereditary and environmental factors. He stated the two generally recognized auditory deficiencies:

1. Early degeneration of the nerves.

2. Otosclerosis in which interplay of heredity and environment can often be traced. This is a progressive deficiency of hearing, usually noted in early adult life.

In any discussion of otosclerosis, two questions must be considered:

1. What local changes seem hereditary, and what is the basic factor causing these changes?

2. Are there environmental factors influencing the occurrence of the condition as a clinical entity or its appearance after what seems to be a period of quiescence? Albrecht, in 1936, believed that for the condition to actually occur, an inciting environmental factor was necessary.

In morphological observations, Wilson stated that the most common site for otosclerosis was in front of the oval window, between the stapes and cochlea, "the site of predilection."

Small at first, it may spread to the adjacent capsule and involve the ligament of the stapes, diminishing hearing efficiency. Islands of cartilage are missing in the labyrinthine capsule. Picture is one of slow absorption.

As to environmental factors, pregnancy in predisposed persons may precipitate it or increase deafness already there. The essayist cited views for morphologic change:

Wittmaack: Damming back of venous blood, in the labyrinthine capsule.

Mayer: Reaction to mechanical influence of smaller vessels and osteoporosis, "mechanical traumatism."

Gray: Degenerative process, whole organ of hearing.

Fraser: Catarrhal otitis or other infections in predisposed persons.

Nager and Meyer: Link to Paget's or other bone disease.

The minute fissula ante fenestram (connecting vestibule and middle ear in fetus) was next discussed. This fissula may not disappear and the normal progress may be interrupted and slow developmental irregularities spread into the surrounding area. In final hypothesis, the author emphasizes that morphologic appearance pointed to alteration in the capillary circulation, considers the trouble in the middle ear a likely source. He considered that it was not justifiable to write down otosclerosis as a degenerative process — it should be considered an inherited weakness, with degrees of susceptibility to environmental factors, internal and external, which may be interactive.

Nager[5] has contributed generously from his research material and in another contribution emphasized the importance of complete study of the bone pathology in order to get the explanation of otosclerosis. In the enchondral or endosteal layer embryonic changes remain, but in the periosteal layer vital bone changes are also seen. Fractures through periosteal layer heal in normal manner; in enchondral layer, bone regeneration is lacking and labyrinthine fistulae occur; Paget's and von Recklinghausen's diseases are discussed. The chief problems of otology, middle and internal ear suppuration, and otosclerosis, are closely linked in the study of petrous pathology.

Researches in calcium balance, vitamin C content found in the urine, and the correlation of parathyroids and generative glands in katabolic changes, were the theme of Seiferth's paper[6] on the etiology and treatment of otosclerosis. He believed that otosclerosis was traceable to hormone and endocrine disturbances; specific directions were given for parathyroid, vitamin C and generative gland hormone administration. Chief success should be obtained in young people with moderate deafness — otosclerotic patients limited to hearing of ordinary conversation, at only a metre's distance, are out of the question.

Adam[7] reported from Hungary on the treatment of otosclerosis, experimentally, by thymus extract in connection with a preparation of calcium. This was administered intramuscularly, using small dosage of a strong extract, 12 injections in all. The rationale of this treatment depended upon the assumption that otosclerosis usually begins and the thymus atrophies at puberty. The author noted that the thymus proliferates and otosclerosis ordinarily improves during pregnancy.

Following in somewhat the same vein as Seiferth, Jung[8] and co-workers reported from the laboratory of clinical surgery at Strassburg on the calcium content in otosclerotic patients. On the assumption that hypocalcemia tends toward a state of parathyroid hypofunction and finding that in 15 cases of otosclerosis studied nine were found below normal, they administered parathyroid extract, calcium, phosphorus and hypophyseal extract. They obtained favorable results in both general and otolaryngological symptomatology. This was a report limited as to numbers and any results which justified analysis as to their therapeutic administration in otosclerosis.

Influenced by the work of Gray, two points seemed to indicate further investigation: *1.* the effect on hearing of introducing fluid into the tympanic cavity by way of the Eustachian tube; and *2.* the variation that occurs in hearing in otosclerosis over a long period during which no treatment is given. Suggit[9] treated 11 patients in this way, seven in both ears, and four in the worst ear only. There was no subjective improvement in any of the 11 cases, but no increase in deafness. Three showed improvement by audiogram, and one improvement in tinnitus.

Injections were given through the Eustachian canal under endoscopic vision. The improvement in the three cases was comparable to that observed in thyroxin injections — an average gain of about 10 dcb.

Radzyminski[10] methodically outlined various symptomatic modifications — surface, physiologic and neurologic — which occurred during pregnancy and childbirth, with particular reference to the hypophysis. Next, he traced the effect of these epochs upon calcium metabolism and the blood ratio. Considering next the endocrines, the author stated that a prin-

cipal and most important relation between these glands and the ears is shown in otosclerosis. He then quoted Escat in the statement that:

1. Otosclerosis concerns the periods when the genitals attain their maturity and when they atrophy.

2. The affection is aggravated in childbirth and lactation.

3. The process of affecting the osseous capsule of the two sides of the internal ear resembles acromegaly, gigantism, osteomalacia, rachitis and other affections provoked by endocrine activity.

4. The process is arrested after menopause or artificial suppression of the menses.

5. Otosclerosis is, in large majority, a female affection.

The author examined 100 pregnant women in the Gynecological Clinic of the University of Poznon, and from these examinations and with record of age, duration of pregnancy, etc., prepared elaborate tables to show the results of caloric and galvanic tests, and diagrammatic curves. The influence upon the ears is threefold:

1. Through contraction of the afferent vessels.

2. Through the permeability of the vessel walls.

3. Through variation in blood pressure.

In closing, the author emphasized the influence of parathyroids, metabolism of calcium, phosphorus and sodium, alkalinization of the blood, and accumulation of the products of decomposition in provoking nervous irritation and special excitability of the static organ.

A paper was also presented by Sommerfeld[11] on otospongiosis (otosclerosis) and the influence of the pregnancy hormones on the osseous capsule of the labyrinth. The author undertook to analyze all the cases of otosclerosis in the consultation service of Prof. Portmann's Clinic, 1928-37.

In their reported series of 58 cases, 50 were female, eight male. He stated that Bezold and Siebenmann had reported one-third men and two-thirds women. He reported a limited

experimentation upon three guinea pigs and was planning further research on a larger scale.

One should not proceed further in the study of otosclerosis without a tribute of appreciation to George Shambaugh, Jr.,[12] for the splendid review of chronic progressive deafness, including otosclerosis and diseases of the inner ear, for 1937, presented under the title of "Progress in Otolaryngology" and published in November, 1938. This review for 1937 has been accurately and precisely collaborated and, of course, should not be duplicated here.

A report of the Otological Research Committee was made by Wilson[13] before the American Otological Society. In the anatomical and pathological investigations, wide anatomical variations were established which occur between the stapes and the cochlea — the seat of predilection for otosclerosis. The most characteristic feature was a minute channel, found only in man, filled with connective tissue, with a rim of cartilage surrounded by the bone of the otic capsule, called the "fissula ante fenestram." The surrounding cartilage varied in thickness, and the bony trabeculae external to it contained narrow spaces of varying extent. In certain cases, masses of cartilage were seen at an age when the entire otic capsule was ossified. This occurred most frequently:

1. Along the cochlear margin of the fissula.

2. In connection with aberrant fissular extension above the tympanic opening.

3. In the fissula at or near its tympanic opening.

4. Where the fissula extends towards a stapes joint.

This cartilage changes to atypical osseous tissue, and possibly later an attempt would be made to resorb and reorganize such areas with resulting sclerotic bone.

In a published report, Dr. Anson was said to have shown that in the endolymphatic duct there were structures present capable of the physiological mechanism of absorption. This might help toward a better understanding of pressure changes in the membranous labyrinth. An effort was made to classify otosclerosis in a recognized general pathological group with such diseases as Paget's, but important basic differences were found.

On the physiological side, Prof. Tait and the English group felt that normal hearing resulted from the vibration of the basilar membrane, and that the hair cells and nerve elements in the cochlea were not essential to the production of the cochlear response.

The opinion of Dr. Davis was "for the present we adhere to our view that both normal hearing and the electrical activity of the cochlea depend upon the integrity of the organ of Corti."

The results on localization of frequencies in the cochlea by making localized lesions in the cochlea at points marked 200 and 250 cycles on his map, were reported by Dr. Cullers.

Prof. Tait, working with the Harvard group, made small lesions in different parts of the cochlea on cats and studied subsequent functional and histologic changes — total deafness in some cases, others only partial, with loss not confined to any one frequency.

In discussing a localized form of Paget's disease with otosclerosis and cataract, and the question of parathyroid insufficiency, Rayband and Guidoni[14] took occasion to discuss the general question of the relation of Paget's disease and von Recklinghausen's disease, which have so much anatomic analogy. This particular case presented Paget's disease of the tibia and slight degree in the skull, associated with painful cramps and continual tinnitus of both ears. Clinical and evolutional differences of the two processes were discussed, and the like analogy elsewhere between acromegaly and gigantism whose syndromes were related to hyperfunction of the hypophysis. No final decision as to the identity appears to have been reached, though the localized Paget's and otosclerosis were emphasized.

Bast,[15] in his report on the "Development of the Otic Capsule; Fossula Post Fenestram," stated that cartilaginous and bony changes in the fossula occur in about 5 per cent of cases. This incidence is much lower than that of similar changes in the "fissula ante fenestram." If such changes are forerunners to the formation of otosclerotic bone, then the observation that otosclerotic bone occurs more commonly anteriorly than posteriorly to the stapes has a developmental basis.

The author stated that types of fissulae described were purely developmental and anatomical, not functional types.

They were more variable and less constant than the fissulae, and occurred in 67 per cent of all ears studied.

Anson, Karabin and Martin[16] reported on "The Stapes, Fissula Ante Fenestram, and Associated Structures in Man" from the embryo of seven weeks to 21 weeks. The authors considered the anatomic study of the ear required as precise information as to form and structure of the stapes as on those of the surrounding area — vestibular window, fissular tract and islands of osseous tissue of types peculiar to the temporal bone (precursor to otosclerosis). The following conclusions were given:

1. The stapedial cartilage was clearly defined in embryo, 22.8 mm.

2. At about 135 mm., embryo of the stapes still cartilaginous, but at 161 mm. centres of ossification had formed.

3. The stapes was relatively far more bulky at this time and was close to attaining adult size.

4. The vestibular window was lined by a rim of cartilage throughout life. In a person aged 70 years the cartilage was not wholly replaced by bone.

This article has been abstracted and reported because of its close connection elsewhere with initial otosclerotic changes.

G. E. Shambaugh, Jr.,[17] under the title, "Recent Advances in the Diagnosis and Treatment of Deafness," stated that the diagnosis of curable from incurable deafness depended to a large degree upon the hearing tests. Certain advances in hearing tests had a practical application in the diagnosis of two important kinds of curable deafness:

1. Secretory otitis media, a form of conductive deafness long known.

2. Exudative labyrinthitis, a form of inner ear or perceptive deafness, first described two years ago.

It was long considered that pure conduction deafness showed a greater loss for low tones than for high tones. The author showed by audiogram a case of otosclerosis with stapes fixation showing pronounced loss for low tones and almost normal hearing for high tones. He compared this with an

audiogram from a patient with secretory otitis media, showing a loss of hearing for high tones as great as in the low tones. That this was not an inner ear lesion was proved after th restoration of normal hearing by evacuation of the fluid from the middle ear, and inflation.

The author offered this explanation for the difference: That low-pitched tones reached the labyrinth through the drum membrane and ossicular chain via the oval window, while high-pitched tones entered through the round window. If this be correct, a traumatic rupture of the drum membrane should give the same test as otosclerosis, whereas a suppurative otitis media should give the same picture as a secretory otitis.

The second type of curable deafness, *viz.*, exudative labyrinthitis, depended for diagnosis upon the demonstration of diplacusis, which is the symptom where a given tone is heard at a different pitch in the two ears. The author discussed the different sound reception in the affected and normal ear in case of diplacusis. Its complete explanation would require almost the quotation of the article in its entirety.

In conclusion, the author stated that diplacusis was not infrequently found with specific research. When associated with low-tone nerve deafness and vertigo, with hearing fluctuating from time to time, the patient probably had an inflammatory lesion in the membranous labyrinth which might be called exudative labyrinthitis, and comparable in etiology, pathology and clinical course to iritis.

The suggestions from this well illustrated article should greatly aid in advanced diagnosis.

Crowe[18] reported on "Diagnosis and Differential Diagnosis of Deafness." He cited three common types of impaired hearing:

1. Lesion of the conductive apparatus which interfered with transmission of the sound to the cochlea.

2. Lesion in Corti's organ or the cochlear nerve which interfered with the perception of sound.

3. Combined conduction and perception deafness.

Ninety-eight per cent of all deafness was due to lesion of the middle or inner ear or cochlear nerve.

In order to cause deafness, tumor of the brain or other central lesion must involve auditory pathways of both sides (except where the cochlear nerve was affected within the temporal bone or cerebellopontile angle).

With each decade of life, high frequency tones were more impaired.

He stated that many abnormal conditions in the middle ear did not impair hearing, *e.g.*, retracted membrane, adhesion to promontory, fibrous or myxomatous tissue in the round-window niche.

These three conditions of the middle ear did impair hearing: *a.* Partial or total obstruction of the Eustachian tube. *b.* Any lesion interfering with movement of the ossicles. *c.* Fixation of the footplate of the stapes from otosclerosis.

For diagnosis and prevention of common deafness in early adult life, one must realize that in children with obstructed Eustachian tubes hearing was more impaired for high than low tones. Such obstruction was caused by: *a.* Overgrowth of pharyngeal lymphoid tissue. *b.* Hypersecretion of tubal mucus. *c.* Chronic suppuration of one or more peritubal cells.

There may be slow development of high-tone deafness from this cause even to age of 10 years — watch with nasopharyngoscope.

Méniére's disease caused progressive deafness. When this was treated by intracranial division of the vestibular nerve, some of the cochlear fibres were occasionally divided, with loss of one or more octaves in the high tone of scale.

Laboratory studies obtained cochlear localization for 2,048 d.v. and higher frequencies, but none lower.

Two important differential diagnoses:

1. With normal Eustachian tube and middle ear, audiogram starting well for low tones but increasing gradually, the lesion suggested is atrophy of the cochlear nerves supplying the basal turn of the cochlea — organ of Corti not affected.

2. With normal Eustachian tube and middle ear, abrupt or localized impairment of high tones in the audiogram, indicate

that both nerve and organ of Corti in the basal coil are atrophic.

Tuning forks and audiometer were insufficient for differential diagnosis. Detailed history, complete upper respiratory examination and use of masking in the tonal study were all necessary.

Mortimer, Wright, Collip and Thomson,[19] in January, 1939, published an article under the title, "Intranasal Administration of Estrogenic Hormones in Constitutional Deafness." In treating cases of atrophic rhinitis with intranasal administration of estrogenic hormones, several cases of progressive deafness and tinnitus reported improvement.

Cranial skiagrams in 70 cases of constitutional deafness paralleled a constitutional background in certain respects similar to that reported in the nasal cases treated. It was suggested that, without direct evidence, there might be an otogenital relationship.

Cases of constitutional deafness have been treated in Montreal in this way since July, 1936 — 94 females and 59 males, at an average of 37 and 39 years of age. The cranial skiagrams of both sexes showed evidence of dyspituitary background, that in the females moving toward the hypofunction level, and in the males to the hyperfunction level.

Most of the patients were ordinary clinic patients diagnosed as otosclerosis or nerve deafness, without attempt at selection. The daily nasal insufflation of 1 cc. oil containing 1,000 international units of estrin was used for at least three months.

Hearing change by the audiometer was quite remarkable in many cases — others continued to regress. Some gained 55 dcb. in some tones, and gains were noted throughout the audiometric range.

After alluding to the stimulation given to bone conduction at the American Otological Society meeting in Detroit in 1936, Coleman[20] discussed the protracted studies of Watson in Prof. Knudsen's laboratory at the University of California with a special induction type of electrodynamic vibrator of wide frequency range: *a.* The cranium was found to vibrate like an elastic spherical shell at 1,000 cycles. *b.* An accurate method of determining the thresholds of bone conduction was devel-

oped. *c.* Bone conduction hearing was most acute with mouth closed, teeth clenched and ear canals closed.

A method for the hearing of speech by bone conduction was also developed by Watson. Their experiments indicated that the hearing of speech by bone conduction might obtain as high a degree of perfection as by the usual method of air conduction.

Under the title of "Recent Progress in Oto-Neurology," Shapiro[21] discussed disturbances in hearing due to central lesions. He reported a case of Lawson's tumor of the midbrain with a hearing loss of 12 inches on the right ear, and 60 per cent on the left, postmortem examination revealing no other cause for deafness. He referred to the opinions of Luttin, Grake and Kompaneets as to contralateral deafness in central lesion. Not much has been reported by American observers, bringing out existence of central deafness. While these deafness cases have a progressive phase, they really concern the internal ear.

Under the general heading of "The Eye and Ear in Industrial and Social Life," Bean[22] discussed the ear involvement. Inasmuch as in the consideration of the ear in industrial workers we think in terms of boilermakers, airplane and cloth weavers' deafness, every industry should strive to eliminate excessive and pathology producing noises. Occupational noises can be divided into two classes: noises of sudden magnitude, such as explosions, causing deafness by immediate effect upon the cells of Corti's canal in the cochlea, with or without middle ear injury and rupture of the drumhead; and second, sounds of prolonged duration, of high pitch and full of overtones, which produce their effect first by stimulation and later degeneration in Corti's organ, first in the ciliated cells and then progressing to nerve fibres.

Tinnitus and vertigo often accompany such defective hearing.

Pathologic proofs of this classification have been based upon animal experiments with guinea pigs.

Pilots of airplanes are subject to three types of deafness: *a.* That due to rupture of the drumhead, as in rapid descent. *b.* That due to continuous sharp, reiterated explosions of the

exhaust, usually temporary. *c.* Temporary deafness affecting the testers under short exposure.

Gottstein and Kayser found marked impairment of hearing in 50 per cent of smiths and machinists. Both found 35 per cent marked deafness in coppersmiths.

Bertoin[23] reported on deafness due to lesions of the labyrinth as sequels to caisson disease. Two cases in workmen were reported. The effect most frequently following decompression was great vertigo, intense noise, and partial or complete loss of hearing. The usual course would be either: *1.* recovery; *2.* partial retrogression in auditory-labyrinthine disturbance; and *3.* permanent depression of the cochlear and vestibular functions. The cases reported came under the last category. Even where the lesions were total, the evolution was progressive. Early hyperacusis is gradually transformed into unilateral deafness and loss of posterior labyrinthine function.

Experiments on the conduction of sound in the ear, principally done with rabbits, were reported by Kobrak.[24] The aim of the experiments was to throw light on two important physiological questions: *1.* what is the function of the acoustic reflex of the muscles of the middle ear; and *2.* what is the mechanism by which they execute their function? In all experimental observations, the diminutions of the vibrations of the ossicles due to muscular contraction is an outstanding finding. The presence of a reflex contraction during strong stimuli and their absence in weak tones point toward a protective function. The acoustic stimulus controls the strength of the muscular contraction very accurately. The muscle contraction in turn changes the sensitivity of the ossicular chain. Each tone has its own characteristic sensitivity of sound conduction. The author makes the interesting comment that it is evident that the ear is not built in accordance with sound recording as developed by science. The middle ear has a resonance frequently located exactly in the area of the spoken voice.

Kobrak, Lindsay, Perlman and Dubner[25] again reported on the effect of limited cochlear lesions, on cochlear potentials and middle ear muscle reflexes. In these experiments a comparative study was made of the effects of perforating lesions of the

cochlea on electric potentials picked up from the cochlea and on the acoustic reflex response of the muscles of the middle ear. This experimentation was prompted by previous widely divergent results. For example, Dworkin found, in 1935, that a small hole drilled in the cochlea would deafen the cat. Bast and Eyster found, in the same year, that destruction of as much as the upper three coils of the guinea pig cochlea did not markedly impair the cochlear response. In the authors' experiments, the acute disturbance with loss of labyrinthine fluid produced little change in the cochlear response but abolished the reflex. This clearly demonstrated the independence of the cochlear response from the hydrostatic conditions within the cochlea. This was compatible with the hypothesis of Lurie and Davis, who believed that electric potentials originated in and are dependent upon the hair cells, the appearance of which, histologically, would give an estimate of electrical activity.

Meleanby[26] discussed the destructive VIIIth nerve changes in dogs under a diet deficient in vitamin A. The overgrowth in bone in the labyrinthine capsule and in the modiolus of the cochlea caused pressure on the fibres of the VIIIth nerve when young animals were brought up on this diet. Bony overgrowth was not limited to the labyrinthine capsule, but other nerves were squeezed by it.

In connection with this histologic study of cochlear conditions, mention might be made of the stereoscopic binocular microscope developed by Tullio[27] for microsurgical auricular study in man and animals; a technical demonstration of procedure, with illustration of apparatus, was presented in the article; eight illustrations and a table for microscopic adjustment were included.

Daikichi Oda[28] reported on the histologic study of 35 ears with marked degree of impaired hearing for all tones with frequencies above 1,024 d.v., and slight or moderate impairment for all the lower tones. Rinné test was positive in them. The evidence from this material as to tonal localization along the basilar membrane was reported as in complete agreement with Crowe, Guild and Polvogt. The area of most importance for 8,192 d.v. was in the middle of the lower basal turn; for 4,096 d.v. in the upper end of the lower basal turn; and for 2,048 d.v. in the lower part of the upper basal turn. Evidence

for lower tone localization did not support the theoretical views that there was even a moderately restricted area of response to these frequencies.

Polvogt[29] has given us a careful contribution on the endolymph. He first reviewed the anatomy of the endolymph closed system, which is so small that it contains but a few drops of endolymph, but is believed to be of prime importance in the normal functioning of the auditory and vestibular apparatus. End-organs in the vestibular system have a blood supply, but no one has demonstrated blood vessels in the end-organs of the cochlear nerve. Anatomic and experimental studies would indicate that a constant secretion of endolymph is necessary for normal hearing, but histologic studies correlated with hearing tests, shortly before death, did not bear this out. The object of this paper was to emphasize the importance of the endolymph in hearing and equilibrium, and the need for investigating the labyrinthine fluid in normal or diseased ears. He called attention to Crowe's suggestion that Méniére's disease might be due to physical and chemical changes in the endolymph. While the deafness involved was of inner ear type, its connection with progressive changes was obvious.

Many points of interest are brought out in the contribution of Kerridge,[30] entitled, "Recent Advantages in Knowledge Concerning Hearing and Speech." There has been little fundamental change in the theory of hearing since the time of Helmholtz. Wever and Bray, using the cathode ray oscillograph, found that the auditory nerve had the property of conducting impulses much more quickly than any other nerve in the body. From this followed the study of what has been named "the electric response of the cochlea." This electric response of the cochlea is a change of electric potential occurring one-tenth of a millisecond after sound is incident on the eardrum. The change in electric potential varies as often as the air on the drumhead, and there is a remarkable resemblance of the wave form of the sound and the wave form of the electrical change. From this point, the author gave a careful description of experimentation involving the mixed effect of the electrical response of the cochlea, and the action potentials of the auditory nerve, under varying conditions of nerve stimulation, traumatism, anesthesia, fatigue, etc., and variant relations from the neighborhood of round and oval windows. This has

an important bearing upon electric registration of hearing, comparative changes in different pitch levels, and study of the mechanism of hearing in different parts of the auditory range. The article closed with a discussion of the nerve connections of the cochlea.

Bone conduction in the experienced pilot and its probable interpretation was studied and reported by Firestone:[31]

1. Bone conduction audiograms were taken on 109 pilots consistently flying from seven to 20 years.

2. The audiograms as a group suggest the audiograms of otosclerosis.

3. Noise and machinery rhythm are other factors.

4. The group studied had 800 to 14,000 hours' flying experience.

5. The author conceived a new otologic entity (aero-otosclerosis).

6. Measures of preventive otology were advised, such as preliminary audiograms, protection against vibration by cushioning, periods of rest from flying, and microscopic studies of the cochlea.

Aubrey, Olivier and Oumicov[32] presented a paper on auricular ionization of choline salts. This was used for chronic suppurative and nonsuppurative cases of deafness with sclerosis, tinnitus and, less often, vertigo. They reported observations on 10 patients; presented two audiogram illustrations. Of these 10, five had very good results, three improvements, and two failures. They recently used a fresh solution of bromide of methylacetylcholine and bromide of bromocholine. It should have special value for vertigo, labyrinthine deafness and tinnitus, provided the latter is recent.

At this point it might not be inappropriate to review the contributions on the impairment of hearing from certain drugs and chemical agents by such trustworthy investigators as Covell, Taylor, Mosher and others. This is not strictly within the field of limitation suggested by the topic, "Progressive Deafness," yet its basic influence may be of great importance, and its prohibitional value greater still.

Covell[33] discussed the effects of drugs on the stria vascularis, and stated that the length of the list of substances affecting hearing is as appalling as the diversity of their natures. Histologic changes in the peripheral auditory mechanism as well as lesions in the central auditory pathways, when correlated with the results of hearing tests on animals, should aid in better understanding of action of such drugs as quinine. The microscopic studies of such vessels as the stria would be worth while to locate vascular stasis, measure its proportion in various turns of the cochlea, and to determine whether the distention of the vessels in the stria vascularis alter the pressure of the intracochlear fluid and so influence hearing. It has been shown that quinine bisulphate and sodium salicylate produced degenerative changes in the cytologic constituents of the cells of the stria. Injections of codeine, soluble barbital and other drugs failed to reveal the same changes in mitochondrial preparations of the animal's cochlea, though codeine produced a distention of the vessels within the stria. So it is probable that many different kinds of agents produce a stria stasis without significant microscopic changes. Vascular stasis within the cochlea would theoretically result in lack of proper nourishment to cochlear structure, and might alter the pressure of the intercochlear fluid. Kobrak found that nicotine produced a tetanic contraction of the tensor tympani muscle, and caused a strong movement of the labyrinthine fluid.

It may finally be possible to associate a loss in hearing with changes in vascularity in the stria.

Another contribution from Covell[34] on the histopathology of the peripheral auditory mechanism in drug-injected animals was published later in the year. This study, result of several years' work, was confined to the histopathologic changes produced by quinine and salicylates because of their common use and frequent resultant deafness. Pregnant guinea pigs which received quinine and salicylate during the period of gestation revealed degenerative changes in the mitochondria of the cells of the stria vascularis, while in the fetal guinea pigs, there were even more pronounced cellular changes. Degenerative changes were found in the external and internal hair cells. The external sulcus cells in these drug-injected animals were found to be swollen and vacuolated, and the endings of the cochlear nerve were involved, even to loss of continuity in the more advanced stages.

Covell and Black[35] studied the maximum value of a cochlear response. They stated that if the hair cells were essential in the production of cochlear response, the maximum value should reflect the physiological condition of the greater number of cells. Previous cytopathologic studies on the effect of quinine and sodium salicylate on the external hair cells revealed a scattering of degenerative cells for the early stages of the drug intoxications. The author discussed the application of readily soluble substances to the round-window membrane, which, however, involved the consideration of several factors, each of which might cause a decrease in response value. The predominating factor was found to be the change in conductivity of the cochlear fluids with the ionization of the agent. The study of response values to sodium chloride, magnesium sulphate, alcohol and other agents has a research rather than a practical value in progressive deafness.

Taylor[36] added to his previous contributions under the title, "Impaired Hearing from Certain Drugs and Chemical Poisons." It has been clinically, experimentally and pathologically demonstrated that some drugs and chemicals have a selective action for the auditory apparatus. They may cause deafness; they may also injure the cortical centre of hearing. Drug idiosyncrasy may be a factor. The permeability of the placenta has permitted the administration of such drugs during pregnancy to endanger the ear of the unborn child. Quinine has been particularly important as it has been so frequently used in the induction of labor. Besides quinine, the salicylates and alcohol have been causative agents, and, less frequently, tobacco, arsenic, lead, phosphorus, carbon monoxide, carbon disulphide, oil of chenopodium, mercury, morphine and the aniline dyes. Drugs and industrial poisons capable of endangering the auditory apparatus should have had greater consideration and more careful study as etiologic factors in deafness.

A careful animal experimentation to show similar changes in the ear of mother and fetus after ingestion of quinine was presented before the Otological Society by Mosher.[37] In all, 54 animals, including six adults used for controls, were studied in this investigation. The initial effect of quinine and salicylic acid, either given in overwhelming doses or doses over protracted periods until the animal became toxic, was hemorrhage. This occurred in any part of the cochlea about the basilar artery or even in the medulla.

The frequency with which hemorrhages in the cochlea occur with or without medication is their worst feature. Only when repeated to the point of formation of fibrous tissue in places vital to tissue, to the strangulation of ganglion cells, or to the obliteration of fundamental blood supply, do they become important as a cause of loss of hearing. The X-ray was used to determine pregnancy and most of the feti were obtained by abdominal section. The average dose for the animals was around 20 gr.

The blood supply of the cochlea and its vulnerability to damage was then discussed.

The action of quinine in both the adult pig and the fetus was to produce congestion of the cerebral vessels, and the vessels of the cochlea, associated with hemorrhage in every location. As a result of the sodium salicylate series, it was the author's impression that, taken as a whole, sodium salicylate produced less cochlear congestion than quinine. The mapharsen experiments were most striking of all. In the fetus, rather than in the mother, large hemorrhages were found in the scala vestibuli, the vestibule, and in one of the semicircular canals.

The writer's experimental animals showed that quinine, sodium salicylate and mapharsen fed to the mother or injected caused hemorrhage in the cochlea of the fetus. What happened in animals probably happens in man.

The influence of quinine in large doses on the labyrinth was reported by Chryssicos and Yanoulis.[38] The authors pointed out that in Greece, quinine tablets were frequently used in attempted suicide. The effect of large doses of quinine was studied during life in 25 and by histopathology in three cases. In 10 of the 28 cases, nothing of interest was noted in the ears. In some of their cases, they noted congestion of the tympani, in one an exacerbation of chronic otitis, two cases of persistent vertigo and instability, and practically all the patients had tinnitus, a sign of labyrinthine congestion. They did not observe hyperacusis, blindness or permanent deafness from the quinine intoxication. There were some cases of spontaneous nystagmus, and caloric tests showed a diminution of excitability of the labyrinth.

Steinberg,[39] in a contribution from Leipzig, discussed inherited eye and ear diseases, with chief attention to the former.

Deafness and dumbness, as well as vestibular inexcitability, were based upon a degeneration of the ganglia cells and, therefore, grounded on central changes. Retinitis, idiocy, deafness and dumbness were not inherited singly. The disturbance of metabolism was inherited and led to an increasing deterioration of the ganglia cells and the many-sided damage which was seen.

Wever[40] reported on the measurement of the width of the basilar membrane in 25 human cochlears at a number of positions from base to apex. By the graphic reconstruction method these positions were determined for each cochlea on terms of the distance from the basal end.

No significant relation was found between the form of the basilar membrane, age, sex and race, or condition of hearing, as shown by audiometric tests made before death. It was pointed out that a theory of hearing that postulates a differentiating rôle for the width of the basilar membrane must allow for a wide range of normal variation in the apical region. Mathematical results were discussed in the article.

Mygind and Dederding[41] tried to demonstrate the influence of water metabolism on hearing, both in dehydration and hyperhydration. These authors previously published some 77 experiments on 46 patients. In 33 of these experiments, indisputable improvement in hearing was noted. In some cases the improvement was accompanied by spontaneous nystagmus, and the improvement, further, had the character of improved sound conduction rather than improved sound perception. They referred to the early work in the late nineteenth century of Politzer and others with pilocarpine treatment, aimed at dehydration. This demonstrated improvement of audition. A most distinct effect was noted with salyrgan (Mersalyl), a mercurial preparation which rapidly produced profuse diuresis.

The authors in carrying out their experiments for hyperhydration required fasting patients to drink 1,000 cc. water early in the morning. Weight and total diuresis were measured. In a number of cases of Méniére's disease, hearing was reduced during the test, in shortening of air and bone conduction and impairment to hearing of whispered and spoken voice. There was also fluctuation in the blood concentration. A most

interesting feature was that the blood concentration curve and the acoustic reaction curve in 77 out of 97 cases ran fairly parallel. Control experiments on otosclerotic patients showed no acoustic reaction, indicating that this depended on a sufficiently mobile stapes.

By hyperdosage of water, Furstenberg and others had provoked giddiness and nystagmus in a Méniére patient. It is true that a diet poor in salt is probably beneficial in Méniéres' disease. From previous experiments the authors concluded that in isolated cases, temporary hearing improvement occurred with increased dosage of sodium chloride and limitation of water.

Baccarani[42] has contributed a 100-page or more article, in three parts, covering the entire field of arterial hypertension syndrome in otorhinolaryngology. In the second portion, the author discussed the relation of cardiovascular pressure to disturbances in hearing, with a classification of different types of tinnitus and vertigo. The circulation and vasomotor innervation of the internal ear was described, with the various relations of arterial sclerosis to cochlear pathology, vascular spasm, vasomotor compensation and disturbance of local circulation.

He noted that on the side of diminished hearing there was frequently a hypoexcitability of the posterior labyrinth in the caloric test. Tinnitus of vascular origin was in many subjects related to local transitory disequilibrium. For circulatory compensation, the author believed it necessary that there be an irritative focus in the auditory complex capable of producing vasomotor changes in predisposed subjects with unstable equilibrium.

This contribution as much concerns internal as progressive deafness.

"Some Problems of Aural Medicine" were reported by Mygind and Dederding.[43] In a long discussion of some problems of aural medicine, they outlined a treatment for deafness in certain cases: *1.* Thorough preliminary examination of habits and surroundings of patient. *2.* Water test performed to determine any eventual retention of water. *3.* A general treatment consisting of dehydration and stimulation of the vasomotor function, avoiding drugs as far as possible.

4. A thirst cure carried out with from 500 to 800 gm. of fluid per day, according to the individual — the diet practically salt-free. *5*. Baths, massage and arc lights used to stimulate capillaries in skin, muscles and lungs to higher capacity. *6*. Ammonium chloride given, 1 gm. t.i.d. for dehydrating effect. *7*. Local treatment consisting of daily Eustachian catheterization. *8*. Results controlled by regular vestibular and cochlear functional tests.

Furstenberg, Crosby and Brownell[44] presented a paper on "Hypertensive Deafness." The purpose of this was to take account of stock of the problem of splanchnicectomy as to technique and success in hypertensive deafness. In a careful review of the anatomy and physiology of the cochlea, they considered it safe, from experimental determinations, to conclude that tones of high pitch were localized in the basal portion of the cochlea. Tones of low pitch were less accurately placed and localized. An elaborate study of the brain tracts was given, which emphasized our limitation of knowledge of the neuro-anatomy of the auditory nerve; and on the theory that resection of the splanchnic nerve terminals would bring about a vasodilatation of the renal vessels, and cause appreciable reduction of blood pressure in hypertension, a technique for splanchnicectomy was organized in the surgical department of the University Hospital at Ann Arbor.

After a statistical study of 50 patients so operated, the following results were reported:

Group 1 (7 per cent): No improvement, neither drop in blood pressure nor relief of symptoms.

Group 2 (16 per cent): Slight improvement. Little drop in blood pressure, but appreciable symptomatic relief.

Group 3 (29 per cent): Moderate improvement. Marked relief of symptoms, and appreciable drop in blood pressure.

Group 4 (33 per cent): Marked improvement. Consistent drop of 40 points or more in systolic blood pressure, and relief of all symptoms.

Group 5 (15 per cent): Cures. Maintained normal blood pressure for at least six months, with complete symptomatic relief.

These results compelled optimism for a possible operative procedure which might arrest the progress of deafness in hypertension, and in some cases bring about improvement, through elimination of tinnitus and throbbing noises in the head.

The "Neurologic Aspects of Temporo-Mandibular Disorders" were discussed by Char.[45] He stated that ear symptoms were a cardinal feature of temporomandibular joint disorders. This might consist of intermittent or continuously impaired hearing, a stuffy feeling in the ears, tinnitus, etc. The Eustachian tube might be compressed by pressure from the condyle of the mandible and trauma occurred in the tympanic structures. Small branches from the auriculotemporal nerve to the tympanic membrane, external canal and tympanic cavity were considered contributing factors to the ear symptoms. Fifty-two of 165 patients were reported as having varying grades of catarrhal deafness. The opening of the bite relieved the obstruction and improved the hearing. Some relief to deafness was obtained from inflation of the Eustachian tube. The VIIIth nerve was not involved, apparently, in these temporomandibular disorders. All otologists did not agree that opening of the dental bite relieved tinnitus and deafness.

MacCready[46] reported a case of inner ear deafness from tetanus antitoxin injection. Following a prophylactic dose of tetanus antitoxin, a general reaction developed with local swelling, which continued for four or five days, including marked dizziness and the sudden development of bilateral deafness. A diagnosis of diffuse serous labyrinthitis was made with hyposensitivity of the semicircular canals, as demonstrated by the caloric test. There was a 45 per cent hearing loss on both sides, which had persisted for a year without progressive change.

McCord, Teal and Witheridge[47] made a report on "Noise and Its Effect on Human Beings." In noisy industrial employments they found groups of workers, below 30 years of age, in which 50 per cent had some degree of impaired hearing.

Audiometer experiments indicated that workmen exposed to particular frequencies after a time became deaf to those frequencies, and in many cases the deficiencies appeared first in the higher range. The lower part of the organ of Corti, concerned with the perception of the higher frequencies of sound, appeared especially vulnerable.

Auditory diseases arising from occupation were generally due to a chronic degeneration of the cochlea. Occupational deafness usually developed in the worker gradually, but in many cases of exposure to intense high frequency the accrued damage was permanent. They reported the findings of Gottstein and Kayser; of 75 smiths and machinists, 40 per cent were definitely hard-of-hearing. Holt reported 10 per cent of coppersmiths with normal hearing, and Barr reported 9 per cent of boilermakers with normal hearing.

In the New York Department of Labor, 246 persons were found deaf; 91 had histories of infectious ear involvement, and in 155 the deafness was traceable to industrial cause. Peyser believed that most of the deafness among locomotive workers was due to bone conduction vibration. McCord reported 52 per cent incidence of occupational deafness in the telephone ear (left ear) of train dispatchers. Loud and sudden noises, explosions and excessive pressure were found particularly dangerous. Noisy work done in confined space was more harmful than that done in the open air. In industry, air conditioning offered little promise of protection against noise for workers employed near the origin of noise. Vibration in ranges below audibility has a prominent rôle in injuries arbitrarily classed as noise diseases.

Sudden temporary deafness due to congestion of the cochlea was the subject of a contribution by Zanzucchi.[48] This was characterized by severe deafness of the cochlear type, of rheumatic origin, following earlier otitis media and associated with severe tinnitus. After leeches and intramuscular injections, symptoms promptly disappeared. The author stated that Citelli had reported three cases of this type in 1926, and two more in 1933. Citelli believed the condition due to a congestion of the cochlea, following first a congestion of the labyrinth. Comparing the present case, the author noted that in his case both sides were affected, and that the hyperemic picture was stopped before permanent deafness, as in Citelli's cases — the latter were unilateral.

Horace Newhart[49] reported on hearing deficiencies in relation to speech defects. Of 50,000,000 children of school age, 40,000 are blind, 60,000 totally deaf, 3,000,000 hard-of-hearing, and 4,000,000 defective in speech. Subnormal hearing caused a very substantial proportion of speech defects. Recent devel-

opments in electroacoustics with the audiometer and electrical hearing aids were demonstrated. Among school children a relatively slight hearing defect may constitute a significant loss. Ears must be tested separately — the better ear must be excluded or masked. Test of relative hearing acuity with air and bone conduction audiometer is essential to diagnosis. Recommendations were made that: *a.* every speech defective child should be given a hearing test, preferably with the 2-A audiometer; *b.* every person directing speech correction should have a satisfactory course in the fundamental principles of the testing of hearing; *c.* every speech department should encourage establishment of lip-reading and make regular periodic hearing tests.

"Deafness in the College Age" was the substance of a communication by Kafka.[50] He noted that college people would wear glasses but were reluctant to wear a hearing aid. The common causes for deafness were outlined as follows:

Acute Infectious Diseases: Scarlet fever, measles, chicken pox, diphtheria, typhoid fever, pneumonia, meningitis and encephalitis.

Hereditary Factors: Deaf-mutism, otosclerosis.

Infected Foci: Teeth, tonsils and adenoids, sinuses and intestinal tract.

Injuries to Auditory Mechanism: Industrial and occupational deafness from noise and vibrations.

He reviewed the systems in large institutions which had no soundproof rooms available, and audiometers in only two locations at that time.

He believed the college student should have a rigid hearing test on entrance, and uniform classification for hearing deficiencies, in order that the student might be helped during initial stages of trouble.

The hearing of speech was discussed by Macfarlan,[51] who pointed out the educational, social and economic importance and particular value in learning a language. He emphasized the standardized use of a phonograph, both for the aid to the otologist in selection of a hearing aid and estimating the degree of disability of hearing that, which the patient is most concerned in hearing.

Firestone[52] added a word of caution under the title, "Volitional Deafness Versus Malingering or Simulation of Deafness" — a comparison of ototerminological values. The writer has found that the term "malingering" was injurious to prestige before any court of justice, and has contributed to miscarriage of justice. His suggestion that the term "volitional deafness" is preferable to "malingering" or "simulated deafness" in otomedicolegal cases appears logical. Volition is one of the normal elements of a normal personality, and the onus of proof that a claimant's interposition of his volition in the normal response to auditory stimuli is abnormal to this particular patient rests upon the attorney for the claimant, and is thus shifted from the otologist. The otologist merely establishes objectively his conclusions. With the term "malingering," the ability of the otologist is apt to be on trial rather than the veracity of the injured workman's claim. This brief extract is interpolated simply as an interesting phase of any type of temporary or chronic deafness, and seems to present a certain value.

Hand[53] reported ear manifestations following head injuries. The most common ear symptom following head injury was nerve deafness and loss of hearing for high tones. The next in order of frequency was labyrinthine vertigo, aggravated by quick head movements. The writer stated that ear symptoms following head injuries may be caused by degenerative changes in the ear nuclei, sluggish labyrinthine circulation, increased intralabyrinthine pressure. The writer urged the careful otologic studies and analysis of vestibular findings. Hyper- and hypoirritability of the labyrinth should both be studied.

In this connection mention should be made of the Pure-Tone Audiometer for School Use, described by Newhart.[54] This had an operating range of 128 to 8,192 cycles, a high grade dynamic receiver, and was designed for the examination of large groups of children, and could be had at a comparatively low cost. A school, college or industrial group could be "screened" at low cost, and then deficiencies in progressive deafness could be checked later to determine exact loss in hearing, and its type.

Hayden[55] discussed "Audiometers and Hearing Aids." In his article, he discussed the principles involved and range of usefulness in audiometers, including maskers, microphone

amplifiers, soundproof rooms, rubber receiving cups and specially prepared charts. He then reviewed the principles of the various hearing aids and the testing of these audiometers.

After testing 30 persons moderately hard-of-hearing by different types of machines, he made the following conclusions:

1. Audiometers, despite the foregoing variations, furnished the best means of testing hearing acuity.

2. Any one of the four tested will be more useful in clinical practice than any other means now available for testing and recording hearing acuity.

3. The lack of purity of sound wave apparently did not introduce any serious errors that were not largely explainable by other causes.

4. A quiet room is essential. The need for a soundproof room increases as the loss of hearing to be treated decreases.

5. An audiometer should be part of the office equipment of every otologist.

Batson[56] has made interesting comments on the perceptive side of deafness, under the title of "Hearing and Deafness." He stated that hearing may be defined as "ability to comprehend speech," and deafness as "interference" with this. Speech comprehension involves, in addition to hearing organ, an intact and agile central nerve mechanism. He discussed the variant attentive faculty in receiving audiometric tests.

Perceptive deafness was found in children, of very irregular character, with perhaps only an island or two of hearing. Such children required special training to learn to speak, preserve their speech, and acquire auxiliary faculties to compensate for hearing loss. The group with active middle ear disease and otosclerosis enjoy electric hearing aids which amplify sounds. To the perceptive deaf patients, increasing the loud sounds is painful. In conclusion, he emphasized that deafened children must be trained for suitable occupations.

Nash[57] presented a very important paper on "What Can Be Done for Chronic Progressive Deafness — A Study in the Treatment of Deafness" in the symposium before the American Otological Society. In this study of 200 cases, the author considered aural pathology leading to progressive deafness

and discussed the conservative treatment of this condition. Patients were observed for five years, tested by spoken, whispered voice and audiometer — the youngest age 5 years and the oldest age 78 years. In types of pathology producing deafness, chronic catarrhal otitis media led by 57.5 per cent of the 200 cases. In the tympanic cavities, normal drumheads were 58 per cent; chronic purulent types, active and inactive, 26 per cent. In condition of the Eustachian tube, 52 per cent were always open. For otologic axioms essential to normal hearing: — external ear must be patent, Eustachian tube normally open, tympanic cavity contain air at atmospheric pressure without fluid behind it, the drumhead normal, air pressure on both sides equal, ossicles mobile, and internal ear mechanism and cerebral centres functioning normally.

The customary routine conservative treatment was followed, including corrections of intranasal pathology, and treatment directed to correct and stimulate nerve mechanism and improve general health. Percentages were given showing gain or loss of hearing under treatment, and in final conclusion the author gave the opinion that submucous resection would not improve the hearing of any of these patients, that present conservative treatment of chronic progressive deafness was unsatisfactory and inefficient.

It has been quite impossible to classify or group some of the immediately foregoing articles, though it has seemed best to abstract and report them as bearing a certain relation to the general subject of progressive deafness. Passing now from the more definitely clinical and research types of contributions to the medical and surgical articles of therapeutic nature, much of the same difficulty and irregularity will be found, as many of these articles have reported experimental procedures.

Group 2, Medical Therapeusis and Systemic Readjustment: Under the title, "The Hearing Problem: A Challenge to Medicine," Kinney[58] made a survey of the whole problem and classified activities in four especially interested groups: *a.* the American Society for the Hard-of-Hearing; *b.* commercial firms which manufacture and sell hearing aids and audiometers; *c.* Federal and State governments; and *d.* the medical profession.

The American Society for the Hard-of-Hearing has as main objectives the prevention of deafness, conservation of hearing

and rehabilitation, and has done much to spread the knowledge of lip-reading.

The commercial firms have made great strides in electrical hearing aids and audiometers for individual and mass testing of hearing ability.

The Federal government made hearing surveys and assisted in the standardization of audiometers by the Federal Bureau of Standards — failing in the former because there was no individual examination of those thought to be defective.

Under medical progress, the author divided the deaf into two groups: *a.* those developing deafness before; and *b.* after age of 21 years. There were approximately 50 per cent in each group. Later life deafness was divided into two groups, 70 per cent either otosclerosis or senile, and 30 per cent catarrhal deafness, with an occasional case of VIIIth nerve tumor.

Early life impairments were divided into three groups: *a.* those occurring before birth; *b.* those occurring as a complication of contagious disease; and *c.* those due to otitis media of nonspecific origin.

The author discussed the various means by which the numbers of these groups could be diminished.

Dworkin[59] discussed the prevention of deafness in Cincinnati school children. The phonograph audiometer was introduced in 1925 by the League for Hard-of-Hearing in co-operation with the Bell Telephone Co. It was claimed that this made practical for the first time the mass testing of school children. By the 4-A audiometer an entire class of 40 children could be tested in about half an hour. The ones with defective hearing were then checked on a 2-A audiometer for tones of different pitch and intensity. Combining the 4.3 per cent positive hearing loss with the 3.2 per cent doubtful hearing loss, there was 7.5 per cent hearing loss of the total of 6,344 school children.

An inquiry questionnaire was sent to families of children with defective hearing in regard to colds, earaches, running ears, etc. The author concluded that the control of upper respiratory infections would lessen the inroads of defective hearing.

Moore,[60] of the Cleveland Clinic, in discussing the treatment of deafness, asserted that there were three objectives in the treatment of the deaf patient: *a.* improvement in hearing; *b.* prevention of further hearing loss; and *c.* development of patient's morale.

The first objective was difficult to obtain. He reported improvement in a certain percentage of cases of nerve deafness with positive Rinné and loss of high tones by inflation of the Eustachian tube, which also helped conductive cases if the tubes were obstructed, but gave no improvement if they were not.

The Eustachian tube orifices should be studied by the nasoscope with great care for adhesions or adenoid tissue, early signs of benign or malignant growth, evidence of chronic infection in the tips of the tube, or pus from diseased sinuses.

A suppurative sinusitis may be responsible for the ear condition, and the writer emphasized the importance of the interference from deviated septum, absorption from infected tonsils and nasal allergy.

He stated that many cases of advanced conductive deafness in later life were due to neglected cold episodes in childhood and early maturity, and advised inflation of the tubes after acute conditions had subsided.

He felt that the most important development in recent years was the periodic testing of school children, and asserted that some degree of deafness had been found in about 13 per cent of the children tested.

In early nerve deafness, all foci of infection should be removed, survey of habits and drugs should be made to detect toxic nerve effects, tobacco and alcohol should be controlled, and the use of quinine guarded. In cases of nerve deafness, the Wassermann reaction should be ascertained.

He discussed subacute and chronic otorrhea and felt that if conservative measures did not stop the discharge, a mastoidectomy should be done.

Kerridge[61] has continued the study of school children in a contribution entitled, "Hearing and Speech in Deaf Children." In a school population of approximately 500,000, about 500

children were given special educational facilities by the London Council for hearing defect; 42.5 per cent of all the cases were deaf from birth, 34 per cent deaf from infection of the ears following infectious diseases and fevers.

The author discussed the air conduction hearing curves in the different types of deafness.

In relation to the cause of deafness, the most common lesion in congenital deaf-mutism was a lesion in the middle ear, although other malformations occurred. High and middle tone maximum losses were present in about equal numbers in the cases deaf from otitis media.

The writer called attention to the fact that true congenital aphasia was rare; that speech is ordinarily learned by hearing and imitation; that the speech of the deaf was rarely normal in sound.

Speech problems to the teacher of the deaf fell into three categories: *a.* to correct minor defects of the partially deaf; *b.* to teach speech and vocabulary to the severely deaf; and *c.* to preserve speech and enlarge vocabulary in those suddenly deaf after normal speech had been acquired.

The author finally discussed the relative amount of hearing loss pertaining to normal and down to increasingly poor speech, with a comment on the continuance of poor speech among older children with a certain percentage of hearing loss in spite of many years of special tuition.

By far the most complete analysis of the education of a deaf and hard-of-hearing child has been presented by Goldstein.[62] The author first discussed congenital deafness, erroneously labeled for decades "deaf-mutism," and defined it as that form of deafness resulting from the transmission at birth of a physical imperfection, a pathologic process affecting the otitic capsule or brain in its embryological development, or some histopathologic element of or in the peripheral or central areas associated with the organ of hearing.

Congenital deafness might be: *1.* biologic, or *2.* pathologic. *Biologic* congenital deafness must have as its basis the development of an imperfect embryo in the growth formation of the otic capsule, temporal bone, brain or histologic structures associated with the mechanism of hearing. There might be

absence or abnormality of essential structures of the temporal bone, a deformed cochlea, histologic deformities of the organ of Corti, absence of neuroglia in the cochlear nerve, under-development of that portion of the brain cortex in the temporal region associated with the hearing centres.

Pathologic congenital deafness includes the transmission of hereditary tendencies to deafness through consanguinity in marriage, syphilis, tuberculosis, rickets, intrauterine hemorrhage, etc.

The author stated that over 30 per cent of reported congenitally deaf cases exhibited a residuum of hearing amenable to re-education and stimulation, and condemned the words semideaf and semimute as without standard accuracy.

Another type of congenital defect is known as congenital aphasia or mechanical inability to produce speech, dependent on retarded development of the speech centre or associated fibres and often found in children with normal hearing.

Inasmuch as this article principally concerns stabilized condition of deafness, even though applicable to those with progressively acquired disability, it is perhaps inappropriate to present in complete detail the careful demonstration and purposes of the acoustic method which the author has presented, as well as standards of measurement of residual hearing. The discussion of tactile impressions and differentiation are particularly illuminating.

The article is concluded with a classification of deafened children and observations as to their training.

A contribution on "The Deaf Child" was presented by Franklin.[63] The recent advances in the physics of sound have found an application in the treatment and investigation of deafness in children, the most striking effect of which is its interference with the normal development of speech. After discussing the social phases of it, the author divided types of deafness into congenital and acquired. In his opinion 80 per cent of such deafness occurred without a history of heredity or pathologic taint. Of the remaining 20 per cent, heredity, consanguinity and syphilis were predominant features.

Acquired deafness was caused by inflammatory, suppurative or toxic invasion of the hearing mechanism. The degree of

deafness depended upon the virulence of the infection and the part of the auditory mechanism involved.

The author described his deaf-mute clinic for the study and aid of the preschool group of deaf children. The staff included an otologist, psychologist and speech therapist. Inasmuch as this falls in the same category as the previous abstract, it is hardly appropriate to go further into detail here into the electroacoustic and electrovibratory apparatus used in the clinic or discuss the progress results among the patients.

Tumarkin[64] has presented an article on "The Prevention or Palliation of Deafness." He first pointed out that the ear and mastoid disease had a greater mortality than typhoid and scarlet fever between 1926 and 1936, and that the greater part of aural disease occurred under the age of 5 years. Steps taken to control middle ear suppuration required: *a.* elimination of predisposing causes; *b.* elimination of accessory causes; and *c.* improvement of the existing treatment of established suppuration.

For the first, the ideal was to eliminate the common cold, scarlet fever, measles, diphtheria, etc. Treatment with serum and toxoid immunization would help. In the accessory factor elimination, diseases of adenoids and paranasal sinuses were emphasized but the author felt that too much tonsil and adenoid work had been done. In reference to suppurative otitis media, the author held that the dry treatment was best with applicators and powder blowers.

A municipal service under charge of a conscientious aurist for the prevention of deafness was outlined.

"The Prevention of Hearing Difficulties in Children; the Rôle of the Family Physician" was the title of a contribution by Stone.[65] He outlined the commoner causes of childhood deafness as follows: *a.* sinusitis; *b.* hypertrophied and diseased tonsils and adenoids; *c.* otitis media complicating pneumonia, measles, scarlet fever, etc.; *d.* foreign bodies in the external canal; *e.* diving and swimming; *f.* traumatic injuries to the ear; *g.* congenital syphilis; *h.* toxic neuritis, quinine, salicylates, etc.; and *i.* systemic diseases, endocrine dyscrasias, allergic diseases, etc.

The treatment fell into three classifications: *a.* prevention; *b.* specific therapy; and *c.* rehabilitation and readjustment to society.

Bradbeer[66] presented an article on "Chronic Deafness," in which he discussed the causes of deafness and types of chronic deafness. He made three main divisions: *a.* chronic catarrhal deafness associated with changes in the middle ear and ossicular chain; *b.* otosclerosis involving change in the bony capsule of the inner ear, one of the unsolved problems of medicine; and *c.* nerve deafness, with its customary background. He reviewed the customary treatments and attention to foci of infection.

Kamizsai[67] reported his technique for the developing of hearing in the deaf-mute. This was based upon the principle of stirring the cortical organ to action by a tone machine acting in the frequencies of speech. These different frequencies of sound stimuli brought about by a tone generator with 12 or more earphones made possible the treatment of a dozen patients at a time. The intensity of the tones was varied from time to time. The treatments took place twice daily, 40 to 60 minutes at a time. His treatment was on the basis of the thesis, that ability to hear was the result of the sound stimuli of the outer world; therefore, he expected an improvement through sound stimuli. The results of this experimental treatment are not at hand.

Miller[68] presented his acoustic method of stimulation under the title of "The Re-education of Auditory Perception." This depended upon the utilization of sound vibration for the stimulation of residual hearing, education and re-education of the hearing mechanism. He cited the work of Archigenes in the first century, Alexander Tralles in the sixth century, Ernaud and Periere in the eighteenth century. Itard, in 1802, cited some success with deaf-mutes; Toynbee, in 1858, used a crude apparatus, and Urbantschitsch followed. The machine presented was an audio-oscillator producing various pitches. An apparatus capable of producing a continuous and varied change of tone with controlled frequency and volume. The audiometer, an essential factor of the machine, was an audio-frequency oscillator, covering the complete range of audio-frequency.

Desjardins[69] wrote on the "Toxic and Therapeutic Action of Roentgen Rays and Radium." The work was mainly a catalogue picture of various technicians in X-ray treatment. The field covered many types of ear trouble, chronic eczema, acute perichondritis, hematomas, otosclerosis, deafness and tinnitus,

tuberculosis and benign tumors. He even reviewed a malignant group. As stated above, this was a cataloging review of the possibilities and scope of this form of treatment, with cautions as to its use and dosage.

Under the title, "On the Origin of the Wever-Bray Response and on an Electrotherapy of the Ear," Kupfer[70] discussed the theory of electrotherapy, pointing out that the two phenomena on which the theory was based were the electroacoustic effect and the Wever-Bray response. Experiments in 1912 furnished striking evidence that hearing must be due to a chemoelectrical phenomenon. The fact that electrical stimulation sets up sensations of sound, whereas the nerve itself is insensitive to sound, suggested the idea that, also, ordinary tone stimuli must necessarily operate by the generation of electrical current impulses. The Wever-Bray response was evidently due to contact and electrokinetic phenomena at the marginal surface of liquids and solids in the cochlea. These contact potentials were probably set up by movements of the stapes. The therapy was highly technical and required a very considerable amount of patience and care.

O'Brien,[71] reporting on the treatment of selected cases of chronic catarrhal deafness by X-rays, after reviewing the recent history, with reports from Beck, Dionisio, Desjardins and others, reported the results in his own practice of 140 cases. The cases in this study were all examined by one otologist and diagnosed as secretory or catarrhal deafness — no audiometer tests were taken; the gain was estimated by spoken voice and whispered voice. These patients were seen and treated between the years 1929 and 1935. Results: 73 cases were improved as to hearing and tinnitus; 65 cases were unchanged; two cases were made worse.

Of the 73 improved cases, 67 had nine treatments to each ear, and six cases received only eight treatments.

The author considered nine treatments should be designated as an accepted course of therapy. Of this group of patients, 52 were between ages 20 and 30 years, and 33 between ages 30 and 40 years.

The author discussed the technique involved, and ventured the observation that as X-rays soften fibrous and scar tissue in other places in the body, it is logical to assume that it can and does do this to fibrous adhesions in the middle ear cavity.

In an article on "The Truth About X-ray Treatment of Deafness," Hays[72] cautioned that the use of X-ray treatment only be attempted in selected cases, and in the majority of cases of deafness was ineffective. In those cases in which definite pathology could be detected by the nasopharyngoscope and Eustachian tubes, he felt that X-ray irradiation might itself improve the hearing or clear up the pathology sufficiently to allow suitable local treatment. The rest of his article was devoted to instruction on the technical management of the two types of treatment.

Hays,[73] in discussing modern treatment for defective hearing required a general physical examination as preliminary and prompt care of all positive factors in the nose and throat, with hospitalization of deafened patients frequently advised. He believed X-ray treatment of the nasopharynx would improve the hearing in selected cases, and that emotional instability and glandular dyscrasias tended to upset the hearing mechanism.

Simon[74] has advised the use of the Eustachian heat bougie in his contribution entitled, "The Treatment of Chronic Catarrhal Deafness with the Eustachian Heat Bougie." This was used with an ordinary Eustachian catheter and came in graduated sizes. The bougie consisted internally of two separate, well insulated circuits: *1.* a fine resistance wire through which a low grade current ran from a control rheostat, furnishing any desired heat up to about 180°; *2.* a circuit with a thermocouple in the distal portion of the bougie, which served to register continuously the temperature of the mucosa with which it was in contact.

The author claimed that the use of the heat bougie would open the Eustachian tube for longer intervals than any other procedure, but would help only those patients who have no scar tissue in the middle ear, constricting movement of the ossicular chain.

Chronic hyperplastic sinusitis and chronic catarrhal deafness seemed to have been aided by this treatment; and direct treatment of the Eustachian tube was of value in many cases of chronic catarrhal otitis media, on the principle that aeration of the middle ear did help some patients.

"The Continuously Open Eustachian Tube" was discussed by George Shambaugh, Jr.[75] He stated that Bezold alone gave a

complete account of the continuously open tube. The purpose of this communication was to call attention to the condition, add other etiologic factors and add a therapeutic measure to the one advised by Bezold. According to Bezold, this occurred only in emaciated patients, *e.g.*, tuberculous, with autophonia and a synchronous roaring sound in breathing. Etiologic factors might be the disappearance of the pad of fat around the Eustachian tube in emaciated patients, the result of atrophy of the mucosa in the mouth of the tube with the contraction of scar tissue, adhesions in fossa of Rosenmüller or destruction of part of the tube mouth by carcinoma. The writer suggested in case of low-pitched tinnitus that the tube be studied, autophonia questioned and ascertain if relief came from strongly sniffing. To Bezold's treatment of salicylic and boric acid powder, the treatment of normal saline injections was added. It is to be assumed that this condition would have some relation to progressive deafness.

Goldstein[76] has reported on the Gray method of thyroxin injection through the tympanic membrane under the title, "Thyroxin Therapy; 42 Cases." The basis of this therapy, as quoted by Goldstein, was to ascertain if by producing active congestion in the minute blood vessels in the organ of hearing in cases of otosclerosis, an improvement in the hearing occurred and thus testing the correctness or incorrectness of the author's (Gray) view of the etiology of the disease as expressed by him in the Dolby lecture of May, 1934. The author stated that the form of approach was empirical without physiological or biological explanation. Quoting Gray, "there is no evidence whatever that a deficiency of the natural hormone of the thyroid gland occurs in otosclerosis and thyroxin was not selected to compensate for any supposed deficiency. It was chosen solely on account of its local action." The author used the official maximum, 1/64 gr. in four drops of warm, sterile, distilled water. The anesthesia was brought about by 15 or 20 drops of anilin oil-cocain solution used in the canal for five minutes, then removed and the canal dried. The point of injection was between the tip of the malleus handle and the posterior rim of the annulus tympanicus. The head was thrown well backward, with the mouth wide open, for three minutes. A clinical diagnosis of otosclerosis was essential before the treatment, made as follows: *1.* Family history of progressive deafness; *2.* audiometric and tuning fork find-

ings of defect in upper and lower tone limits; *3.* negative Rinné; *4.* patulous Eustachian tubes; *5.* normal drum membrane; *6.* tinnitus; *7.* paracusis Willisii; *8.* progressive decline in hearing.

The thyroxin treatment consisted of four consecutive injections made alternately in the right and left ears at an interval of one week; preferably upon patients under 30 years of age. The author reported his results in three groups: *Group 1*, no improvement, 9 cases. *Group 2*, improvement with small decibel change, with recession, 10 cases. *Group 3*, continued improvement, 5 to 50 dcb., 23 cases.

Maggiorotti[77] contributed a paper on the use of large doses of sodium glycerophosphate in the treatment of otospongiosis (otosclerosis), a preliminary report. Intramuscular injection was given of 10 to 15 cc. of 15 per cent solution, according to Rinaldi's method, in the three cases treated. Apparently considerable improvement was noted in hearing and diminution of tinnitus. The three cases were observed only a brief period, too short for definite conclusions. Its success was believed to be in the modification of metabolism of calcium and phosphorus. The author's reported results sufficiently encouraged him to urge further experiments on a larger series of cases.

"The Endocrine Therapy; Its Relation to Ophthalmology and Otolaryngology" was the subject of a contribution by Preston.[78] On the basis that hypoactivity of the thyroid gland was frequently associated with symptoms of fullness in the ears, tinnitus or vertigo, and with deafness simulating otosclerosis, thyroid gland extract was given, and many of these patients did well upon it. Insufficient detail was given upon which to base conclusions.

Koch[79] contributed an article upon the Use of Sex Hormones in the Therapy of Deafness. Following the increased investigation in internal secretions, preparations from the male and female germ glands were administered in the hope of aiding hearing by stepping up old age and relieving tinnitus aurium. Twelve patients were treated in this group and elaborate tables have been prepared giving their statistics. Treatments were given upon the following hypothesis — deafness appearing in old age must be subject to dysfunction and poor glandular activity. If substitute glandular treatment be given upon

a symptomatic basis, the administering of sex gland hormones would be justified. In the management of deafness of old age, the use of hormones is justified because harmful results of the hormone treatment should be negative, and apparently a favorable influence resulted upon pathologic conditions in the inner ear. Favorable therapeutic results were the diminution of subjective tinnitus and demonstrable improvement in hearing. Further research, according to the author, is indicated to give greater clarity to this treatment.

Nelson[80] published an article on "Aid to the Hard-of-Hearing," in which he discussed the child percentages with notable loss of hearing in one ear, and the losses which amounted to a handicap. He stated that the audiometer had been of great value in obtaining hearing records, and in aiding the proper recommendation of treatment or mechanical aid. He analyzed the vowel and consonant sounds in relation to their cycles' coefficient, and reported on the decibel loss in which vowels, consonants and conversation could still be heard. This information should be of value to those investigating hearing abilities in different stages of progressive deafness.

Glas[81] reported a new method, "Lavage and Spray of the Antrum, and Problems in Therapy of Deafness." The author first referred to a communication from Whiteman, an Australian physician, outlining this treatment a year before. In brief, this reported a hospitalized patient for 14 days under menthol inhalations for the last 48 hours. Then the maxillary sinus was opened beneath the turbinate, and washed with a catheter with normal salt solution once daily for 14 days. Menthol inhalations were continued. It was stated that 91 per cent of the patients under this treatment, later carried on by the patient once a week, were markedly improved; though it was claimed that at least 70 per cent of the hearing was shown improved, exact statistics were not at hand to corroborate this.

Crowe and Guild,[82] under the title, "Impaired Hearing for High Tones," pointed out that middle ear lesions resulting from occlusion of the pharyngeal orifice of the Eustachian tube, usually caused greater impairment for high than for low tones. In 20 cases, after operative removal of adenoid hyperplasia, they found that limited and carefully screened use of radium emanations at the mouth of the tube produced excellent results. The permanence of hearing improvement cannot yet be predicted.

Group 3, Surgical Procedures: In the symposium before the American Otological Society on "Treatment of Chronic Progressive Deafness," Walker[83] discussed the surgery of the naso-pharynx. After elimination of cases of decreased hearing due to suppuration of the middle ear, only two groups are left:

1. Otosclerosis.

2. Chronic adhesive inflammation of the middle ear due to nasopharyngeal and other infections.

The author cited the address of Emerson on the question of whether chronic progressive deafness was an otologic or rhinologic factor as to etiology. In this paper, Dr. Emerson emphasized his theories of infection occurring from tonsils, accessory sinuses and obstructed nasal drainage.

The author discussed the general anatomy of the nose and throat, importance of the common drainage area of the middle meatus, importance of normal nasal breathing and development of the antra, upper jaw and teeth, and the freedom from septal and turbinate obstruction — all of which were important in relation to the subject. "To prevent chronic progressive deafness, we should do all in our power to insure normal nasal breathing under as natural conditions as possible by hygienic measures and surgical elimination of obstruction." The following conclusions were given:

1. Chronic progressive deafness is due to pathological changes in the middle ear, and coincident atrophic changes in the organ of Corti, etc.

2. The primary cause of this condition is deficient and abnormal nasal breathing.

3. Prevention and relief of chronic progressive deafness depends on how early and how thoroughly obstructions to nasal breathing can be eliminated.

Hughson[84] reported on the rationale and technique, with case reports and observations, of grafts in the round window. The author contended that an exhaustive study should be made of a case of deafness before any operative procedure be attempted. In view of recent findings, involvement of the neuromechanism need not necessarily be a contradiction to operation; the extent

of nerve involvement should be studied by routine loudness balances, repeated bone conduction audiograms with masking, and fatigue tests; advanced age and impairment above 50 dcb. for critical frequency range should probably be considered contrary indications to successful surgery.

The side to be operated on should be partly determined by the bone conduction conditions of the two ears; principal regard should be given to the critical frequencies, 256 to 4,096; a complete clinical survey of the cardiovascular system, glandular system, blood chemistry and serology, nutritional and allergic disturbances is an essential part of diagnostic study.

Under technique, the writer cautioned as to control of bleeding, cleansing of ear canals, liberal incision in the drumhead, careful placement of the graft and brilliant working illumination, with careful follow-up audiograms at frequent later intervals.

Twenty-five operations for blocking the round-window niche with a tissue graft were reported as a treatment for deafness; 18 of the cases have been seen postoperatively for three months, except two with a one-month level. Twenty months was the longest period of observation. In no case was the hearing impaired by operation. Two cases showed no improvement, but all the others did. One patient gained 20 per cent. The average change showed an increase of approximately 10 dcb. in all critical frequencies.

Holmgren,[85] under the title of "Correspondence (Addressed to the Secretary of the American Otological Society)," states that it is possible to obtain permanently open fistulae of the labyrinth, but so far in his own and Sourdille's cases such has been the case only occasionally. The following methods of reaching this goal were suggested: *1.* Opening a fistula by means of lasting pressure; fistulae caused by cholesteatomas have remained open in a fairly large percentage. *2.* It is known that many metals are apt to prevent bone regeneration from their electrolytic reaction. *3.* Radium and mesotorium in suitable dosage have been used successfully. *4.* A second fistula, after closure of the first, as suggested by Sourdille, was another possibility.

The author reports that Prof. Nager, in Zurich, is making histological investigations of the osteogenic changes at the site of the fistula.

This correspondence was in lieu of his intended presentation of technique in various conditions attendant upon the operation, which was unfortunately prevented.

"Otosclerosis, Surgical Therapy; Death of the Patient of Intercurrent Disease; Preliminary Report" was the title of a report by Holmgren.[86] This operation was done in 1936 for a marked reduction in hearing. The operation itself was apparently successful, but nephrolithiasis developed and the patient died finally from embolism in the pulmonary arteries. This was simply presented as a report of a death under the fistulization operation but which occurred from an extraneous cause.

Lempert[87] published a comprehensive article on the "Improvement of Hearing in Cases of Otosclerosis. A New One-stage Surgical Technique." The author referred to the efforts of Jenkins, Barany, Holmgren and Sourdille to ameliorate deafness in otosclerosis by surgical means which led to the making of a new fenestrum in the bony capsule of the labyrinth and preventing its early closure by bony degeneration. The author offered a very careful technique, urging that every step be patiently and skillfully executed in minute detail. He stated that the endaural, antauricular approach to the temporal bone is employed for the following reasons:

1. The tympanocutaneous membrane for covering the trough-like fenestra could be obtained in no other way.

2. The least amount must be sacrificed in reaching the objective.

3. Better visibility and accessibility was obtained.

4. This approach represented the least inconvenience to patients.

5. The cosmetic after-result was best.

The general indications for the operation were: fixation of the stapes; loss of air conduction hearing of not more than 60 per cent under ordinary conditions; normal hearing by bone conduction obtained by masking, or a loss not exceeding 30 dcb. for 512, 1,024 and 2,048 frequencies; absence of suppuration in middle ear and a normal tympanic membrane, without perforation; a normal state of health and a healthy external auditory canal.

The technique in this article was carefully illustrated in the following surgical steps: *1.* The creation of a mobile endaural window for the antauricular surgical approach. *2.* The exposure of the horizontal external semicircular canal. *3.* Plastic construction of a cutaneous tympanomeato membrane. *4.* Liberation of the membrana tympani from the ossicular chain. *5.* Decompression of the dura in the epitympanic recess. *6.* Excavation of trough-like fenestra in external horizontal semicircular canal. *7.* Final liberation of tympanomeatal cutaneous membrane. *8.* Molding of tympanomeatal-cutaneous membrane. *9.* Inserting the above in the trough-like fenestram of the external semircircular canal.

The author maintained that in the 23 patients operated upon by that date, 22 fistulae had remained open. The author believed that a fistula which has not shown signs of beginning regeneration of bone after two months would remain permanently open.

Sourdille[88] wrote under the title, "The Present Position of the Surgical Treatment of Otosclerosis." Two bases for operative procedure for otosclerosis were suggested: *a.* the supposed pathogenesis of the disease; and *b.* acoustic results of the labyrinthine fenestration.

The procedures depending upon pathogenesis were the so-called Wittmaack-Heyninz-Rollin method, designed to combat perilabyrinthine venous stasis (the author called it the elevation of the supratympanic dura mater), and also the Alonso-Chiarino method of removing the left parathyroid or suppressing its function by ligation of the principal vessels, in order to reduce glandular secretion. The procedures based upon the acoustic results of labyrinthine fenestration were cited in the work of Barany, Jenkins and Holmgren.

Sourdille, in 1929, reported on the possible permanent benefit to hearing from an opening in the external semicircular canal, in an operation which he called tympanolabyrinthopexy, which he has steadily improved in the last eight years. His presentation was of an operation completed in three stages with four or five months elapsing between stages. In the first stage, a large flap was cut and released from the outer two-thirds of the posterosuperior wall of the external auditory meatus. In the second stage, the mastoid cells were opened,

the aditus exposed, and the head of the malleus was removed, with care taken to avoid dislocation of the incus. The post-operative aim was for epithelization, as after a radical mastoid operation. The result was to be a large, free tympano-labyrinthine vestibule made of the external auditory meatus plus the mastoid diverticulum. In the third stage, the horizontal semicircular canal was opened with great care to avoid injury to the delicate membranous canal. The cicatricial tissue obtained in Stage 2, called the "plastique interne," was applied to cover the opening and was considered as an extension of the tympanic membrane as a sound amplifier.

The remainder of the article was particularly devoted to a comparison of the Sourdille and Holmgren methods. As to indications, the operation should only be done on primary otosclerosis with ankylosis of the stapes (negative Rinné and prolonged Schwabach), and without too much delay. A very dense mastoid cortex is a contraindication.

Sourdille[89] again wrote on the "Surgical Treatment of Deafness from Otosclerosis." He believed that the mobilization of the perilymph of a filled labyrinth was possible. He considered that it was sufficient to place thin cushions of fat, prosthesis of certain weight, such as a piece of gutta percha, upon the labyrinthine fistula; that these masses in contact with the bony walls of the mastoid received sound vibrations by bone conduction and are displaced by them. This movement induced slight shocks at the level of the labyrinthine fistula and set the perilymph in motion. He believed that indirect mobilization of the perilymph in a filled labyrinth could be produced by mobilization of the membrane covering the labyrinthine fistula by means of a reconstructed tympanic system. His tympano-labyrinthopexy consisted in joining the covering membranes of the labyrinthine fistula with a superior border of the tympanic membrane, the excursions of which were increased by the resection of the head of the malleus.

His operation was performed in three principal stages: the first two devoted to the transformation of tympanic system, and modifications of the mastoid region; the third to the establishment of the labyrinthine fistula at the level of the new tympanic system.

His operation had been performed on 109 patients with a result of 10 times or more the previous hearing distance in

40 per cent, five to 10 times previous hearing distance in 14 per cent, mediocre results of two to five times previous hearing distance in 20 per cent. There were no fatalities.

From the *Medical News of California*,[90] quoted by the *Journal of the American Medical Association*, is reported a campaign against deafness. California has been divided into 11 geographical areas with a district chairman, each of whom operates under a supervising State director. This was an extension of the educational campaign to prevent impairment of hearing and has been put under way in the State for five years.

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APPLIED BIOCHEMISTRY IN THE ETIOLOGY AND TREATMENT OF CLINICAL CONDITIONS OF THE NASAL ACCESSORY SINUSES.*

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Introduction: On Oct. 2, 1923, the late Dr. J. A. Stucky, of Lexington, Ky., presented a paper before the Cincinnati Oto-Laryngological Society which carried the title, "Suggestions as to the Etiology and Treatment of Nasal Accessory Sinuses."

The opening paragraphs of this paper presented the following: "The object of this paper is to emphasize the nonsurgical treatment of inflammatory or suppurative conditions of the nasal accessory sinuses, whether they be acute or subacute. The contention made by me in the last 15 or 20 years before this and other medical societies, that many painful and diseased conditions of this region are local manifestations of systemic conditions and more satisfactorily amenable to systemic treatment alone or combined with mild and nonirritant local treatment has been verified in numbers of cases in private practice and public health clinics.

"In the last 10 years I have rarely had to surgically drain an emphyma unless the lining membrane of the cavity involved had become pathological to such an extent that it was pyogenic or gave evidence of undergoing polypoid degeneration.

"Localized characteristic pain in the ethmoid frontal, maxillary or mastoid region with a Roentgenogram showing a deep shadow confirmed by transillumination, the anterior and posterior rhinoscopic examination revealing mucopurulent or purulent discharge in the middle meatus, these positive clinical indications do not, in the majority of cases, call for either local or general anesthesia for surgical intervention, either by puncture, irrigation and aspiration, or the well known and accepted more radical procedures."

*Read at a meeting of the New England Oto-Laryngological Society, Boston, Nov. 15, 1938.

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The foregoing represents the school of thought in which I was trained by the late Dr. Stucky. He willed me his library in order that I might be better able to carry on. This oto-laryngological pathway which he taught me has led to a study of biochemistry, and it is the application of biochemistry in the etiology and treatment of acute or subacute inflammation of the accessory nasal sinuses that I wish to present at this time.

CLINICAL OBSERVATIONS.

If one who has a low tolerance for starches starts eating them after a period of low starch intake, he will first observe a gelatinous lump appearing from time to time in the naso-pharynx, which may be removed by forcibly inspiring through the nose and hawking and spitting. In the morning on awakening he will find it necessary to spend a minute or two in clearing the nasopharynx of this gelatinous discharge. If he continues his increased intake of starches, this gelatinous lump will change to mucopurulent in character.

In addition to the necessity of clearing the nasopharynx frequently by hawking, it now becomes necessary to frequently blow the nose. The discharge blown onto the handkerchief causes the handkerchief to stiffen when dry. If the intake of starches is lowered or given up, this nasal discharge first disappears, to be followed by the disappearance of the naso-pharyngeal discharge. Mucopurulent discharge in the nose may be made to appear, disappear and reappear in accordance with a variation in the amount of the daily intake of starchy foods.

If one who has a low tolerance for sugar, especially the type of sugar known as sucrose, represented by white sugar, brown sugar, maple sugar and maple syrup, starts eating it he will soon cross his line of tolerance for sugar and develop a watery discharge from his nose. First, a drop of water gathers at the tip of the nose. As soon as this is wiped away another appears in a short time. The nose is blown frequently but still the watery discharge continues to appear, varying in amount as the intake of sugar varies. Individuals who develop this watery discharge from the nose are able, as a rule, to handle sugars called monosaccharides, such as honey represents, but develop a watery discharge from the nose when

they try to handle sugars called disaccharides, represented by white sugar, brown sugar, maple sugar and maple syrup. In other words, the monosaccharides, glucose, fructose and galactose, are handled well, while the disaccharides, sucrose, lactose and maltose, give trouble and are the ones which easily produce the watery discharge from the nose.

If an individual continues to cross his line of tolerance for both the starch and sugar divisions of carbohydrates, he is very apt to show an increase in the size of lymphoid tissue in the upper respiratory tract. The turbinates within the nose will first increase in size. Following the increase in size of the turbinates, islands of lymphoid tissue appear on the posterior pharyngeal wall, and associated with these islands of lymphoid tissue will generally be found prominent lymphatic bands running up each side of the posterior pharyngeal wall, just posterior to each posterior tonsil pillar. The tonsils, if present, may also participate in this increase in size of lymphoid tissue.

As one continues to observe and study patients, all the above may apparently take place without the patient developing serious symptoms, but sooner or later a block in the body process of cell oxidation takes place, and with the appearance of this block symptoms appear which warn the individual that all is not well with his body, for he no longer enjoys reasonable daily health. The block in the body process of cell oxidation denies his body the glucose and glycogen it needs. For the time being, he is like an individual on a very low carbohydrate diet.

FUNDAMENTALS OF APPLIED BIOCHEMISTRY.

When having clinical contact with a patient there are a number of fundamentals of applied biochemistry that need to be kept in mind if one thinks in terms of the cell and intercellular fluid.

1. Is the condition of the respiratory apparatus such that the intake of oxygen by the body is satisfactory?

2. Is the patient taking into the body the right kind of carbohydrate in order to furnish a kind of carbon that can be oxidized by the cells of the body?

3. Is there a proper intake of organic minerals, especially the so-called oxidizing minerals, iodine, iron, copper, manganese and arsenic, in the food intake in order to create anew oxidizing enzymes within the cell?

4. Is waste material being produced by the body in satisfactory amounts? If so, is it promptly removed from the body?

The answers to these four questions enable one to answer the question: Is there present a block in the body process of cell oxidation? If such a block is present, continued good health cannot be maintained. In due time, infestation of the body by micro-organisms will change into infection of the body by these micro-organisms, or before this takes place there may appear the clinical picture referred to as allergy.

PRIMARY SYMPTOMS OF A BLOCK IN CELL OXIDATION.

The primary symptoms of a block in the body process of cell oxidation are as follows:

1. A more or less constant feeling of fatigue in varying degrees.

2. Inability to sleep well at night.

3. Headaches of the migraine type.

4. Vertigo.

5. Inability to concentrate mentally. Difficulty in making business decisions. Has the butterfly type of mind, which flits from one subject to another.

6. There is a depressed mental state, with discouragement. A feeling that something is going to happen but does not know what it is.

7. Unexplained crying spells are present.

8. Twitching of the eyelids and at times a twitching of the corner of the mouth.

9. On account of the development of accommodation troubles while reading, the use of the eyes for close work is interfered with.

10. Double vision, especially when physically tired. This interferes with driving a car on long trips.

11. The speaking or singing voice tires easily, with loss of resonance.

12. There is shortness of breath of mild degree on moderate exertion.

13. Cramps or pains in muscles of the body most often used. This includes the muscles of the feet, calves of the legs, small of the back, pyloric opening of the stomach, back and upper part of the neck, arms and region of the heart, following effort.

14. Neuritis-like pains in various parts of the body.

15. Tissue soreness in various parts of the body at different times. Patient, for example, will complain of soreness on swallowing, but on examination the throat appears to be negative. The soreness apparently is in the muscles used in the act of swallowing.

This block in the body process of cell oxidation syndrome represents the preclinical state of a number of clinical conditions.

This syndrome may be made to disappear by changing the type of carbon to be oxidized by the body cells and securing an intake of minerals that will insure replacement of the oxidizing enzymes of the cell.

As a rule, each individual does not have all the symptoms of the syndrome, but has a majority of them. Quite frequently one finds an individual who has them all.

KINDS OF CARBON.

Let us consider for a few moments the carbon brought to the cell for oxidation. From the point of view of energy, there are two extreme types of human beings. One of these I call the Plus Family Pattern, because the majority of the collected clinical data is above the accepted normal. The other I call the Minus Family Pattern, because a majority of the collected clinical data is below the accepted normal. The Minus Family Pattern type is easily fatigued and quickly discouraged, and is a low energy type. It contrasts sharply with the Plus Fam-

ily Pattern type, which is indefatigable and undiscourageable; the high energy type moving on to its goal because its dynamic voltage can function to overcome obstacles. In between these two family patterns there is a mixed or intermediate type, which has periods of various amounts of energy, alternating with periods of inertia.

Most common of all are the Minus Family Pattern type, who complain perpetually of being depleted; of being fatigued, morning, noon and night, even after a night's rest, or after the week-end rest from labor. They are the people who have that always tired feeling. When combined with irritability, these sufferers from fatigue are put down by themselves, their friends, or even their physicians, as neurasthenics.

Energy of the body and mind is fundamentally a matter of the carbon metabolism and, in particular, of the sugar metabolism. In seeking to understand these two family patterns, it is necessary to understand the peculiarities of sugar chemistry. As a result of such an analysis, it becomes possible to control the production and output of energy with results appreciated by the individual.

Fat and sugar are the important carbon-containing foods. Because they are the carbon-containing foods, they are the energy-providing foods. For their carbon is burned slowly to form carbon dioxide with energy set free as a by-product.

Of the two carbon-containing foods, fat and sugar, sugar is the more easily burned because it is naturally more easily oxidized. That is, it takes less oxygen to set sugar on fire; less of a draft, as it were. In consequence, sugar or carbohydrate is the preferred food for quick energy production, while fat is the food utilized for slower energy mobilization.

It requires a quick fire to start a slower flame. It takes more quickly flaming paper, wood or oil to start coal burning. As a result it has become an axiom that "fat burns in the fire of carbohydrates." If a body has trouble in burning sugar, it will have difficulty in burning fat. The amendment to this statement is that all individuals are not able to burn all varieties of carbohydrates. A selection has to be made from the list of available carbohydrates. Given a proper selection of carbohydrate, the carbohydrate flame will burn brightly.

A block in the body process of cell oxidation may be brought about by a number of foods belonging to the carbohydrate class. The first of these is wheat. This means that foods made from white flour, whole wheat flour, graham flour, buckwheat flour, also cereals made from wheat, should be omitted from the daily food intake. Rye flour, cornmeal, and oatmeal flour foods are handled very well and should replace foods made from wheat. They do not give evidence of bringing about a block in the body process of cell oxidation.

A second carbohydrate that produces a block in the body process of cell oxidation is represented by white sugar and brown sugar, which are classified as representing a good source of sucrose. This means that white sugar and brown sugar should be omitted from the daily food intake. Honey and the sugar found in grapes and grape juice are handled very well and should replace white and brown sugar.

A third member of the carbohydrate class that readily produces a block in the body process of cell oxidation is citric acid, found in oranges, grapefruit, pineapples and lemons. Citric acid, we read, is oxidized in the body, but in the face of a weak oxidizing mechanism it is oxidized in the body with difficulty, if oxidized at all. If these citrous juices are taken they need to be diluted with an equal amount of water, and then not taken longer than two weeks at a time. When an individual has crossed his line of tolerance for citric acid, he develops a watery discharge from his nose. Malic acid seems to be handled much better than citric acid. Bananas and apples are a good source of malic acid. Apple juice, sold in cans at the grocery store, should frequently replace citrous juices.

OXIDIZING MINERALS.

The oxidizing enzymes of the cells must be constantly renewed, and the oxidizing minerals taken in with the food in organic form appear to be the logical source of the raw material needed to renew these enzymes.

The four best oxidizing minerals are iodine, iron, copper and manganese. I would add arsenic to this group, making the group five in number. That they are true oxidizers is evident from the increase in energy that comes to the individual fol-

lowing their use in very small doses. The size of the dose is important if one wishes the oxidizing effect. It must be small.

Outside of the body the relation of minerals to oxidation of sugar is easily demonstrated. A lump of domino-shaped sugar can be made to burn when a mineral ash is added. Powder from the black portion of a burnt match, cigarette ashes, wood ashes from the fireplace, powdered spinach, celery, banana, kelp or paprika, if added to one end of a domino-shaped lump of sugar will enable a lump of domino-shaped sugar to burn when a lighted match is held under the end that has been dipped in one of the above.

The mineral ash in a substance is of distinct assistance in oxidation and ordinary burning. If fireplace ashes are sprinkled over wood placed in the fireplace, the wood will ignite at a lower temperature. There is no ash from refined white sugar.

Sugar in the blood with a low blood mineral content burns with difficulty, but add minerals, from kelp for example, and the ease of transferring oxygen to the substance to be burned enables oxidation to take place. The painter has added red oxide of lead to start and speed up the process of oxidation. By this oxidation it is possible to accomplish in four days what three to four weeks would require. Apparently a mineral ash must be present to burn glucose. The best results are obtained when the minerals are organically combined.

The rate at which the blood sugar is burned depends upon the mineral content of the blood. The American diet is a very low mineral ash diet.

In the mineral content of the food intake we are dealing with two thoughts. First, there is a total amount of the minerals and, second, the character of the minerals. In ordering the wood for the building of a house one does not order just wood. Building experience has shown that hemlock for the joists, pine for the floors, chestnut for the woodwork and cedar for the shingles work best. In ordering, one specifies the kind of wood and the amount. So it is with the food mineral intake for the body. One does not order simply an increased mineral intake, but for the burning of sugar specifies that the increased mineral intake will especially include, iron, iodine, copper, manganese and arsenic, which are the best oxidizing minerals.

TISSUES AFFECTED BY A BLOCK IN CELL OXIDATION.

As applied body chemistry is used more and more in solving daily clinical problems, and the results of removing the block in the body process of cell oxidation are observed, there comes a time when it is realized that a block in the body process of cell oxidation manifests itself, particularly by the development of clinical conditions in tissues we have come to refer to as epiblastic. These epiblastic tissues developed from the ectoderm are the outer layers of the skin and its appendages, such as the hair and nails; sweat, sebaceous and mammary glands; nervous system, particularly the sympathetic division of the autonomic nervous system; the external sense organs, such as the eye and ear, and the mucous membrane of the mouth, nose, sinuses and anus.

The beneficial nutritional influence on these epiblastic tissues of removal of the block in cell oxidation appears to be responsible for the clinical improvement seen.

EXAMINATION OF PATIENT.

In examining a patient who complains of symptoms suggestive of paranasal sinus involvement, one endeavors to determine whether a block in the body process of cell oxidation is present. If it is present, then, on examination of the nose, one would expect to find a watery or mucopurulent discharge present in varying amounts, the turbinates somewhat increased in size, and the sinuses cloudy or dark on transillumination.

The throat when examined is very apt to show the presence of lymphoid bands on either side of the posterior pharyngeal wall, just posterior to each posterior tonsil pillar, and a number of islands of lymphoid tissue on the posterior pharyngeal wall. The presence of lymphoid tissue depends to a certain extent upon the length of time the block in cell oxidation has existed, and the severity of this block.

Having decided a block in the body process of cell oxidation exists, one proceeds on the basis that this block is primary in the cause of the acute or subacute condition of the sinus, and the part played by the micro-organism is secondary.

TREATMENT.

Having decided a block in the body process of cell oxidation exists, treatment is divided into parts. Part one seeks to

restrict the daily intake of food that is apt to bring about this oxidation block, while part two presents oxidizing catalysts or activators to the body to enable the body to overcome the block in cell oxidation and relieve the carbohydrate intoxication which allows infestation of the body by micro-organisms to change into infection by these same micro-organisms. In treating the patient, he does not as a rule go to bed.

In applying part one of the treatment relating to a restriction of food most responsible for a block in the body process of cell oxidation, the patient is asked to make the following exchanges: *1.* All wheat bread, cereal and other wheat foods are exchanged for rye bread, cornmeal foods and oatmeal flour products. *2.* White sugar is exchanged for honey. *3.* Citric acid juices, such as orange juice, grapefruit juice, lemon juice and pineapple juice, are exchanged for apple juice or grape juice. *4.* Exchange muscle meats for seafood, fish or liver. *5.* Use milk as a food and not as a beverage.

In applying part two of the treatment relating to oxidizing catalysts or activators used to overcome the block in the body process of cell oxidation, I have found three unit doses of plain insulin subcutaneously and three drops of Amend's solution of iodine[1] to be most helpful. The three units of plain insulin are given subcutaneously once each day whenever the patient has an appointment at the office. No effort is made to time the giving of the insulin. The three drops of Amend's solution of iodine are givin in a glass of water 20 minutes before each meal.

Approaching acute inflammation of the accessory nasal sinuses from the viewpoint of a block in the body process of cell oxidation and applying treatment directed towards overcoming this oxidation block, it is usually the rule to have an acute sinusitis last three days. On the second day one may expect the patient to state the pain is 50 per cent less, the nose open and discharge less. The antrum or frontal sinus will be found to often transilluminate 50 per cent better than when the patient was first seen. On the third day there will be freedom from pain and scanty discharge. The antrum or frontal sinus will generally transilluminate in a nearly normal way.

If the inflammation of the sinuses is subacute it will require 10 days to two weeks of treatment to restore the sinuses to

normal. When the sinusitis is chronic in character, from six months to one year will be required to overcome the local pathology. The three units of plain insulin are then given once a week after the first two weeks, and the Amend's solution of iodine in a three-drop dose is given once a day, 20 minutes before one meal. The food restrictions outlined in part one of treatment are also continued. The above treatment is also of value in an acute exacerbation of a chronic sinusitis, enabling one to limit the acute exacerbation to from three days to one week. No local treatment is given the nose. There is, of course, no objection to mild, nonirritant local treatment if one wishes to use such treatment.

CASE REPORTS.

Case 1: C. L., male, age 32 years. One week ago developed a head cold. Two days ago developed pain in both cheeks and forehead. When pain appeared, discharge from nose lessened. During last night forehead pained so much that mustard plaster was applied to forehead. Examination of intranasal mucous membrane showed it to be minus one degree pale in each side of the nose. Right frontal and left antrum failed to transilluminate. Mucopurulent discharge present in middle turbinate region, both sides of nose. Examination of throat showed several islands of lymphoid tissue present on posterior pharyngeal wall and prominent lymphatic bands on posterior pharyngeal wall just posterior to each posterior tonsil pillar. Temperature, 98.8°; pulse, 68; respiration, 18.

Diagnosis: Acute sinusitis due to a block in the body process of cell oxidation. No nasal treatment was given. In order to overcome the block in cell oxidation the patient was asked to exchange wheat foods for rye, cornmeal or oatmeal foods. Omit all foods representing a source of citric acid, such as orange juice, grapefruit juice, lemon juice and pineapple juice. Exchange white sugar for honey. Use milk as a food but not as a beverage. While patient was in the office gave three units of plain insulin subcutaneously in arm, followed by three drops of Amend's solution of iodine in a glass of water by mouth. For home use patient was directed to take three drops of Amend's solution of iodine in a glass of water 20 minutes before each meal. Following day reported at office, stating pain had diminished 50 per cent. Has only a dull

ache in left side of face now. No pain in either frontal sinus region. Nose open now. The nasal discharge has increased 25 per cent. Right frontal and left antrum transilluminate 50 per cent better. At this office visit three units of regular insulin were given subcutaneously in arm. Next day patient returned to office, stating pain in face was no longer present. Sinuses transilluminate alike. Very little discharge from nose now. Lymphoid islands and lateral lymphatic bands no longer seen on posterior pharyngeal wall, showing by their disappearance that the block in cell oxidation has been overcome to a degree sufficient to cause the clinical condition to disappear. The Amend's solution of iodine was continued once a day before meals for one week. At the end of this time the nose was free from discharge. The change in food selection was to be continued indefinitely.

Case 2: Mrs. D. M., age 45 years. Present head cold has lasted three weeks. Nose is now stuffy only at night. Four men's handkerchiefs are used each day in order to take care of discharge from the nose. This head cold is accompanied by daily pain in cheeks and forehead. Wishes relief from continued daily pain in sinus regions. Examination of the intranasal mucous membrane showed it to be minus one degree pale in each side of the nose. Both frontals and antra transilluminate well. Mucopurulent discharge present in middle turbinate region on both sides of nose. No islands of lymphoid tissue present on posterior pharyngeal wall, showing by their absence that the block in cell oxidation has been intermittent or recent. Temperature, 99.2°; pulse, 87; respiration, 22; block pressure, 130/80.

Diagnosis: Subacute sinusitis due to continued block in cell oxidation. No nasal treatment was given. In order to overcome the block in the body process of cell oxidation, the patient was asked to exchange wheat foods for rye, cornmeal or oatmeal foods. Omit all foods representing a source of citric acid, such as orange juice, grapefruit juice, lemon juice and pineapple juice. Exchange white sugar for honey. Use milk as a food but not as a beverage. While patient was in the office gave three units of plain insulin subcutaneously in arm, followed by three drops of Amend's solution of iodine in a glass of water by mouth. Insulin when given in three-unit doses is considered an oxidizing catalyst or activator. For home use patient was directed to take three drops of Amend's

solution of iodine in a glass of water 20 minutes before each meal. Patient came to office each day for seven days, receiving three units of plain insulin subcutaneously in the arm at each visit. At the end of seven days she was symptom-free. The discharge from the nose gradually lessened and disappeared, the Amend's solution of iodine before each meal being continued for one week after the last office visit.

SUMMARY.

1. Many clinical conditions of the nasal accessory sinuses depend on an altered biochemistry of the body.

2. There are definite fundamentals of applied biochemistry.

3. The altered biochemistry is a block in the body process of cell oxidation.

4. This block in cell oxidation is accompanied by a definite syndrome.

5. A block in cell oxidation manifests itself particularly by the development of clinical conditions in epiblastic tissues.

6. Treatment of patient is divided into two parts. Part one seeks to restrict the daily intake of food that is apt to bring about this oxidation block. Part two presents oxidizing catalysts or activators to the body to enable it to overcome the block in cell oxidation.

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107 North Main Street.

PHARYNGEAL BURSAE (THORNWALDT'S BURSAE); REPORT OF 64 CASES.*†

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The presence of pharyngeal bursae is certainly not so rare as one is led to believe by the scarcity of clinical case reports. This is verified by the fact that 64 cases have been observed in the private and dispensary services of Duke Hospital in the past six years.

Historical: Historically, pharyngeal bursae, often called nasopharyngeal bursae, were observed by Mayer, in 1840, and Luschka in 1868. These observations were made, however, in anatomical rooms; and it was Thornwaldt who first attributed clinical symptoms to nasopharyngeal bursae in 1885. Since then the disease frequently bears his name. Most of the literature dealing with the subject appeared in anatomical rather than clinical journals until quite recently. For a complete background of this condition one is referred to the anatomical and embryological articles of Snook,[1] Huber[2] and Dorrance,[3] or the recent clinical reports of Roy,[4] Kully[5] and Woodward.[6] Kully reported 88 cases of nasopharyngeal abscesses over a six-year period of time, a large number of which were due to a typical Thornwaldt's bursa. Yankauer[7] in 1929 reported 155 cases of nasopharyngeal abscesses, none of which he attributed to a pharyngeal bursa, but to a coalescence of adenoid tissue in the region of the median recess, forming a cyst.

Etiology: There had been considerable disagreement as to the origin of the nasopharyngeal bursa, but it now seems to be definitely decided that a pouch develops in early embryonic life, which may persist after birth. Such persistence seems to depend upon the continued union of the pharyngeal segment of the notochord with the pharyngeal entoderm during the period of degeneration of the notochord. This activity occurs over the basilar process of the occipital bone. Pharyngeal

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bursae are reported in 40 per cent of human embryos, but they are certainly observed in much less than 4 per cent in adult life.

Types: The persistent sac, called nasopharyngeal bursa, lies beneath the adenoid tissue and empties by a short duct at the lower portion of this tissue. The sac extends up and back into the periosteum. The bursa may be manifest clinically in two forms: the cyst type and the crust type. These two types vary only in that: in the cyst type the duct is occluded, and in the crust type the duct is patent and discharge is adequate. Apparently the sac does not become infected or cause any symptoms whatever so long as there has been no trauma, such as must occur in the scraping of adenoid tissue. In the greater portion of our cases, clinical symptoms and the presence of a bursa were noted mainly in those cases where adenoids had been removed. The light scraping for adenoid tissue may, of course, not injure the sac opening; thus, no infection would ensue. There probably are many cases of patent bursae that are never suspected because of lack of symptoms.

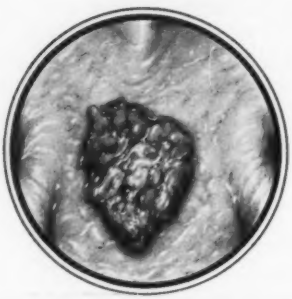
The cyst type of pharyngeal bursa may be a single or multilobular swelling. It is quite possible that patients who present a cyst on one examination may show no cyst during the next examination because of spontaneous emptying by way of the duct. No evidence of spontaneous rupture of the cystic sac was ever obtained. Invariably these cysts are filled with a jelly-like, clear or turbid substance, rather typical of all other cystic contents.

The crust type of pharyngeal bursa may present no evident pathology on examination, especially after a crust has just loosened and fallen into the pharynx. A very careful search may have to be made to find the pin-point duct orifice when no crust is seen. Ordinarily, patients with the crusts notice a bad odor and bad taste in the mouth. The crusts fall into the pharynx anywhere from one to seven days apart. These crusts invariably are conical in shape and facet into the conical depression at the duct orifice, which is located at the lower border of the adenoid area.

Symptoms: The symptoms in both types of disease are practically the same — frequent upper respiratory infections; postnasal discharge — a rather thick and tenacious material

being most commonly described; headaches, which are usually low occipital in type, daily or rather frequent in occurrence; and in several instances slight fever, especially after any attempt at local treatment of the region. In some cases where crusts formed there was occasionally a sensation of dull ache located in the nasopharynx. Eustachian tube obstruction and referred pain to the ear were commonly found.

Complications which may be attributed to the disease are entirely too numerous to mention and are typical of any other focal infection. Outstanding among the cases observed was one of rheumatic fever, and several cases each of asthma,



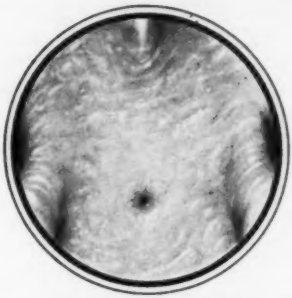


Fig. 1. Crust in nasopharynx. The crust forms as discharge dries after emerging from the orifice of nasopharyngeal sac.

Fig. 2. Appearance of nasopharynx with crust removed. Note pin-point opening of duct in the depression. This opening leads to the nasopharyngeal cyst, which runs up into the periosteum.

arthritis and bronchitis. Various allergic manifestations and eye disorders were also found.

Bacteriology: Cultures were made in all operative and practically all nonoperative cases; but, unfortunately, the reports, at the time of the preparation of this paper, could be found in only 28 cases. Hemolytic staphylococcus aureus was found in 19 cases, beta hemolytic streptococci in six. The other cultures varied from strains of streptococci and staphylococci to one instance each of Friedlander's bacillus and diphtheroids. Mixed infections were commonly found.

Pathology: In all operative cases, specimens obtained failed to show a definite cystic sac, but did show in each instance

infected, scarred and, in most instances, necrotic tissue. In some cases there was a gush of purulent material, which was intermingled with jelly-like material, as the cyst ruptured during the curetting. Kully was able to report a definite cyst wall in some of his cases. An interesting observation was made by Kully in describing all cysts with a smooth surface as originating in the nasopharyngeal bursa, while cysts — usually smaller — surrounded by adenoids were classified as retention cysts. In our group none of the latter type was seen. He divided cysts in general into five groups: first, the retention cysts of the adenoid recess, similar to the cases reported by Yankauer. These cysts are lined with ciliated, cuboidal or squamous-cell epithelium. The second group were those cysts of the mucous glands, which he considered to be true retention cysts and usually due to surgical trauma, obliterating the excretory duct of the mucous glands. These cysts are usually rather small. The third type is the retention cyst (or Thornwaldt's bursa), herein described. Fourth, the degenerative cyst, in which there is necrosis of the adenoid tissue itself and in which the cyst cavity is lined with adenoid tissue. Fifth, the branchiogenic cysts, which are very rare and usually lie in the region of the Eustachian cushions.

Diagnosis: The diagnosis of the pharyngeal bursa can be made in some instances by history from the patient. (Paranasal sinus infection must, of course, be nonexistent.) There is always a postnasal discharge of thick, tenacious mucus, which in most instances the patient describes as clear and jelly-like, or a history which entails the appearance of a crust that drops into the pharynx at intervals of one to seven or more days. The crusts are usually fetid-odored and yellowish or green in color, always conical in shape. Headaches, usually low occipital in type, may be the sole complaint. The headaches at times are really described as being in the region of the nape of the neck, below the occipital region, and at times one would suspect cervical arthritis.

The diagnosis is confirmed in all cases by the reflected image through the nasopharyngoscope. This image reveals the true condition present without distortion. We have not found the pharyngeal mirror to be of much assistance. On one occasion only was it necessary to search any length of time for a bursa, and in that instance it was due to the extremely low

duct orifice in the nasopharynx. The cysts resemble any other type of cyst, and there may be a few dilated blood vessels on the surface. They usually are in the mid-line but may vary slightly and be multilobulated. Occasionally, and especially when the nasal spaces are large and vision is not otherwise impaired, one can see the crust or cyst on nasal inspection with the head mirror.

Treatment: As for treatment, Woodward has suggested biting off the anterior wall of the bursa. The late Dunbar Roy had suggested local applications only, but admitted his results were rather disappointing. In our earlier experience with

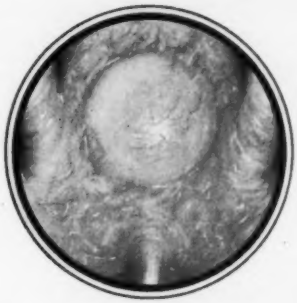


Fig. 3. Nasopharyngeal cyst, due to obstruction of duct orifice.
Fig. 4. Same as Fig. 3 as seen in sagittal section. Arrow denotes location of duct.

this disease we attempted treatments which were not at all successful. We had applied silver nitrate locally, as well as numerous antiseptics and other medications. Even deep X-ray therapy had been tried without success. It occurred to us that good results probably would not be obtained unless the entire cystic sac was removed. Eventually, therefore, we attempted the complete removal of the sac by a thorough curetting of the entire adenoid area. The curetting necessitates much more force than is ordinarily used during adenoidectomy. We have found the Barnhill adenoid curette to be the most useful instrument for this procedure. The anesthesia for curetting the nasopharynx need last only five minutes, or thereabouts.

We have found light ether inhalation anesthesia or evipal intravenous anesthesia to be most satisfactory.

No postoperative treatment is necessary. The area requires much longer to heal than for the usual adenoid operation, due to the presence of scar tissue formed following the previous adenoid operation, and also because deep curetting into the periosteum requires much longer for the regenerating mucous membrane to cover the denuded surface.

Results: In all of the 25 cases operated upon by thorough curetting there was complete recovery of local symptoms in the nasopharynx. The general health of each patient was improved, especially those cases having asthma, arthritis and other systemic complications. We have had no complications following the nasopharyngeal curetting. One wonders, however, why periostitis or other more serious complications have not developed.

Analyses of Cases: For purposes of further study of our 64 cases, they have been divided into two groups — 25 in which operation was performed, and 39 in which operation has not been done to date.

Operative Cases: Of the 25 cases observed and followed by operation, 14 were males and 11 females; 23 were white, and two were colored, with an average age of 26 years. In 21 of the 25 cases, tonsils and adenoids had previously been removed — in two instances operation had been done twice. The period of time following the tonsil and adenoid operation varied from five months to 20 years previously. Six of these cases presented the crust type, and 19 the cystic type. In all of the crust type, tonsils and adenoids had been removed. In 17 of the 25 in which culture reports were available, only one instance of beta hemolytic streptococcus infection was found, that being the one of rheumatic fever and heart disease. The remainder, except for two cases, showed the various staphylococci, hemolytic as well as nonhemolytic. The two exceptional cases were reported as showing Friedlander's bacillus and "no growth."

Nonoperative Cases: Of the 39 nonoperative cases, 16 were males, and 23 females; 38 white, and one colored, with an average age of 35 years. Several patients between the ages of

50 and 60 years brought the average for this group rather higher than for the other group. Twenty-seven showed the cyst type of disease, and 12 the crust type. Twenty-eight previously had the tonsils and adenoids removed, but for 11 there had been no operation. Crusts were found in two patients not having the tonsils and adenoids removed, and in 10 where they had been removed. The organisms predominant in this group were equally divided between beta hemolytic streptococci and the various staphylococci.

THORNWALDT'S BURSA (64 CASES)

25 OPERATIVE		39 NON-OPERATIVE
26	AVERAGE AGE	35
14	MALE	16
11	FEMALE	23
23	WHITE	38
2	COLORED	1
19	CYST (46 CASES)	27
6	CRUST (18 CASES)	12
6	YOUNGEST	5
50	OLDEST	68

YES 21 (2 TWICE)-NO. 4	PREVIOUS T & A	YES 28-NO. 11
5 MO. — 20 YRS.	KNOWN DURATION	1 MO.-19 YRS.
1 BETA HEM. STREP	BACTERIOLOGY	EQUALLY DIVIDED
1 DIPHTHEROID		BETWEEN
1 FRIEDLANDER		STAPHS. AND
OTHERS STAPHS. (HEM. AND NON-HEM.)		STREPS. (HEM. AND NON-HEM.)

Fig. 5. Table of data regarding 64 cases of nasopharyngeal bursae.

Report of Two Individual Cases: It is of sufficient interest to report one or two cases very briefly.

Case 1: Cyst Type: A boy, now age 14 years, had the tonsils and adenoids removed at age 7 years. Aside from the frequent upper respiratory infections of mild severity, and allergy in the family (but not evident in this patient), the history was negative. In early 1936 he developed rheumatic heart disease while living temporarily in New York City. He previously had lived in North Carolina his entire life. He was sent to Porto Rico for the winter months. On his return to North Carolina this past summer, he was found to have a

tremendous-sized nasopharyngeal bursa. Operation was done and pure cultures of beta hemolytic streptococci were obtained. A recent thorough examination, including electrocardiograph, showed no residue of rheumatic heart disease, and this winter it was not necessary to go to a warmer climate.

Case 2: Crust Type: A physician's wife had occipital headaches so severe that medications of all types rarely relieved her. No other cause could be found to account for the headaches. Crusts had dropped from the nasopharynx at intervals of seven days for 10 or more years. Operation completely relieved her headaches and, incidentally, relieved frequent attacks of bronchitis from which she suffered. In addition, a gain of 10 pounds in weight was noted. Staphylococcus aureus was the organism grown in cultures from the bursa.

Summary: In summary, the presence of the pharyngeal bursa, frequently called Thornwaldt's bursa, is not so uncommon as one would suspect. The condition is predominant in the white race, and more often in the well-to-do class. The disease is due to the persistence of an embryological structure which, apparently, lies dormant in most instances until trauma occurs — for instance, adenoidectomy. The cystic cavity, which runs up and back into the periosteum of the occipital bone, may then become infected. If drainage is adequate, the discharge may dry as it emerges from the cystic orifice and form a crust. On the other hand, if the orifice becomes occluded by any means, a truly infected cyst occurs. Apparently these cysts spontaneously drain at times, for one may find them of different size and shape during a series of examinations. The history is rather suggestive, however, in all of these cases, there being a thick, tenacious, jelly-like postnasal discharge, or by the appearance of a crust, dropping into the pharynx at intervals of several days. The infected sac behaves as any other focal infection.

The infection is due to various strains of streptococci and staphylococci, more often the hemolytic type than the nonhemolytic type. The diagnosis can be made from the history alone in numerous instances, and is easily confirmed by the use of a nasopharyngoscope. The treatment found to be most successful in 25 operative cases is the thorough and deep scraping into the periosteum of the adenoid region, by which means every vestige of the cystic cavity is removed. No after-

treatment is necessary, and results have been uniformly excellent.

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ACUTE PSEUDOMEMBRANOUS INFECTIONS INVOLVING THE EYE, MOUTH, THROAT AND URETHRA.*

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Membranous and pseudomembranous infections of the mouth are quite frequently seen in the treatment of nose and throat diseases. Yet there is surprisingly little reference to be found either in textbooks or in the special literature. These cases are found more frequently in childhood and, therefore, we find that the subject is discussed mainly by the pediatrician, who stresses his own aspect of the case. The dermatologist and the dentist also treat many such cases; they, likewise, discuss the subject from their own point of view. The majority of these cases are not of very serious nature and generally clear up in a short time, which perhaps explains why this subject is neglected in our own field.

The first thorough discussion of this subject was made by Fraenkel,[1] who, in 1888, discussed and clarified the histopathology of this type of case and gave to it the name of "Maculofibronous Stomatitis." In our opinion, the book of Prinz and Greenbaum[2] has the most thorough discussion published in recent times. They make the following division, based upon the histopathologic examination of Fraenkel:

"Maculofibronous stomatitis (aphtae)."

A. Acute aphthae: *a.* Habitual and solitary type. *b.* Infectious type.

B. Chronic intermittent recurrent aphthae (Mikulicz).

C. Pterygoid erosions and ulcerations.

We restrict our discussion to the "acute infectious type of stomatitis"; the case reported upon here being, we believe, unique in the literature. Whereas, cases have been reported in which not only was there involvement of the mouth but also some involvement on the face or in the vulva, we report the

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case of a young male with involvement spreading to the eye, mouth, nose, throat and the urethra simultaneously. Neuman[3] reports a case of a girl, age 26 years, admitted to his hospital with an involvement of the eye, mucosa of the mouth and the vulva. He found the upper lid of the right eye swollen and congested, but with no membrane formation. The patient had a membranous involvement of the oromucosa and these same membranous patches were found in the mucosa of the vulva. He called this condition "Vulvitis Aphthosa." Some involvement of the thighs and lower leg were also found. This case, which was very severe, ran a temperature as high as 103° to 104° and cleared up only after intensive treatment of four weeks' duration.

Christlieb[4] had a similar case also. There was present fever, involvement of the mouth, vulva, and the upper part of the thigh. Moro[5] reported a case in which not only was there involvement of the mouth but also of the face and the upper part of the breast.

The following is a report of our case: F. H., male, white, age 23 years, came to the Otolaryngology Clinic of the New York Post-Graduate Hospital on March 4, 1938, giving the following history:

Two weeks previous to his appearance in the clinic he had, as he thought, a severe cold. A few days later his eyes became inflamed and he went to the eye department of one of the large specialized hospitals in New York. He was given black drops (apparently argyrol) to apply to the eyes, and he was advised to bathe his eyes in warm boric acid solution. A few days later he began to feel pain while eating and noticed that sores appeared on his lips, mouth, and in his throat. As a result of this, he went back to the clinic and he was given a mouth wash. The condition became aggravated and he ran a temperature. Then he came to the Otolaryngology Clinic of this hospital on the above-mentioned date. On admission he presented the following picture:

Eyes: The eyes were inflamed and red. Veins on both upper and lower lids were markedly congested. A pseudomembrane on both lids was present, especially on the right eye, which was able to be removed easily, leaving a slightly bloody space. The sight was not impaired. There was an increased lacrimation from both eyes.

Nose: There was increased discharge from both nostrils, which were markedly congested and swollen, but no membrane could be visualized.

Mouth: Lips were swollen and covered with dry, reddish-black crusts, which when removed bled easily, especially in the corners of the mouth. The mucus of the entire mouth was covered with a membranous substance mostly found on the buccae and the palate, but could also be seen on the anterior pillars, and scattered patches appeared on the post-pharyngeal wall and the epiglottis. The tongue was involved

Fig. 1. The patient's appearance upon admission to the clinic on March 4, 1938.

also. The gingiva was swollen and congested, and bled easily on touch. There was a strong, foul odor from the mouth. The submandibular glands and the glands around the sternocleidomastoideus were swollen and sensitive. The membrane, which in some parts of the mouth formed large patches, was easily removable and left a bloody space.

Urethra: The opening of the urethra was quite red and adherent. Urination was painful. On the preputium were a few small, superficial ulcers which were covered by a grayish substance which was removable. Around the glans penis and

in the fold was apparent, also, a grayish membranous substance which did not bleed when removed. No membrane was seen in the urethra.

Laboratory Reports: Blood Count: Hemoglobin, 75 per cent; red blood cells, 4,160,000; leukocytes, 16,700; polys., 80 per cent; small lymphocytes, 16 per cent.

Urine essentially negative.

Smear from the eye showed many pus cells, but no bacteria.

Culture from the eye showed few colonies of staphylococcus albus and diphtheroids.

Fig. 2. Lesions as seen on the lateral wall of the mucous membrane of the mouth and lips.

Smear from the mouth negative for Vincent's disease and gonorrhea, but showed many pus cells, a few Gram-positive cocci in pairs, occasional clumps of small Gram-negative rods, and Gram-positive cocci.

Culture from the mouth showed streptococcus hemolyticus; alpha streptococcus, green colonies predominating; a small area of hemolysis; negative for diphtheria.

Smear from the urethra showed negative for gonococci; no pus cells; a moderate amount of epithelial cells and spermatozoa; a few Gram-positive cocci.

Pathological examination of the removed membrane gave no specific findings.

Blood count, cultures and smears were repeated but the findings remained almost identical. Alpha streptococcus, two types, were constantly the predominating organism in the cultures.

As the condition improved, the hemoglobin increased and the leukocytes decreased. Temperature, 100.8°; blood pressure, 130/90; pulse, 120. Medical examination failed to show any pathology. The patient is not a smoker and used no drugs or strong mouth wash. He was in perfect health previous to this illness.

Fig. 3. Lesions on the soft and hard palate, lips and tongue.

Treatment was as follows: Two per cent watery solution of Pyoktanin was applied many times a day. In addition, notwithstanding the microscopic report, Fowler solution plus glycerine and Vin ipecac in equal parts were given for swabbing the mouth a few times a day, due to the suspected presence of the spirillae. He was advised to eat nothing spicy, nor to have anything too hot or too cold. A neutral mouth wash was prescribed, containing bicarbonate of soda and acid acetyl salicyl. The patient was also advised to refrain from having contact with any member of the family, and to take precautions not to spread the infection.

The patient was observed every second day. He improved rapidly, and after 10 days almost the entire condition had

disappeared, with the exception of the corners of the mouth. A few days later the entire condition cleared up.

Etiology: Fraenkel in his work thought that a case of this type was due to a staphylococcus infection; however, other authors maintained that this condition was due to a diplococcus or streptococcus infection. Lewis[6] calls it "streptococcus pharyngitis" and attributes the condition to streptococci. Flusser's[7] observations were limited only to children; consequently, he found it to be very rare in adults. In his opinion, there is a certain vitamin deficiency which is mainly responsible for the disease. He did not go into the discussion of the bacterium present in these cases. Bonin[8] contradicts Flusser's statements in his report of 30 cases, of which 22 were adults. He believes that the majority of the cases are caused by streptococcus, but he could not show this in all his cases.

Hiller[9] reports five adult cases. We, too, observed a few cases in adult patients. Although none of the authors state specifically that an organism causes the disease, the other symptoms, such as elevation of temperature present in most of the cases, and increased leukocyte count generally found, indicate an infectious nature. Some authors believe that lues affects these cases, but the majority of writers deny this.

Symptomatology: The case herein reported, like many others, had a fever as high as 102° and acted, in general, as an acute infectious disease. Although many of the cases have been found to have a temperature, Hiller did not observe any in his five cases.

While in most of the cases the findings are similar to those reported above with reference to the local symptoms, there are few which involve any mucous membranes outside of the mouth. The involvement in the mouth shows numerous small eyelets of fibronous, sharp, defined patches located, for the most part, on the anterior portion of the mouth and tongue. It may extend to the throat also, and even reach the epiglottis occasionally. Not all cases have swollen lips, but if they are they are generally covered with dry crusts. The gums are inflamed, swollen and bleed easily. Because of the decomposition of blood, there is a strong, foul odor and increased salivation. The adjoining lymph glands swell also. Similar involvement may be found around the mouth or on the face.

Chewing is quite painful, and sometimes almost impossible; hence, nourishment is very often refused, especially by children, causing a secondary emaciation. An abnormal blood count is not characteristic. Bonin found a normal blood count in almost all cases which he reported.

Pathology: Prinz and Greenbaum make the following statement based upon the examination of Fraenkel's work: "The superficial epithelium becomes macerated, and beneath it a fibronous exudate collects, which is firmly attached to the underlying mucosa. Vesicles are never formed. The epithelium becomes necrosed and is cast off, leaving the mucosa covered by a thin formation of epithelium. True ulcerations do not occur. The entire process is somewhat analogous to the formation of the pseudodiphtheric membrane superimposed upon the mucosa."

Differential Diagnosis: Differential diagnosis is not difficult if all of the prevailing symptoms were taken carefully into consideration. First, the possibility of diphtheria and Vincent's angina should be eliminated. The diphtheric membrane generally appears paler and more difficult to remove, but there is a subsequent bleeding aso; therefore, the microscopic examination of the smear and a culture should be the deciding factor.

Vincent's angina appears generally as a single lesion, confined either to the tonsils or to the gums. It is a thick, dirty, gray slough which tends to break into a deep ulcer. Mixed infection which occurs occasionally may mask the true nature of the condition; therefore, a smear should be taken in every case. Agranuleukocytic angina, which is similar to the Vincent, may appear as a single or multiple lesion; however, the patient's condition is very much worse; *i.e.*, it is toxic. In cases of this type, the blood count (Leukopenia) is very characteristic.

Habitual ulcerative stomatitis, or cancre sore, generally appears as a single lesion, without a general inflammation of the entire mucosa. These ulcers may be a few millimetres deep and are undermined with irregular rands, especially at the area where the mucous membrane is above the bony frame, as on the mandibula. Thrush in children is not to be confused with the infectious aphthae. The membrane is lighter in color and very easily removed, but the microscopic examination

sometimes is necessary to prove the origin of the condition. Burns caused by chemicals, especially if they are extensive, may likewise be mistaken. Lues, tuberculosis and cancer, although entirely different, should be still considered. Herpes of the mucosa of the mouth should be excluded.

The prognosis is good under proper treatment.

Treatment: Because of the inflamed mucous membrane of the mouth, mastication is very painful and, therefore, the majority of patients, especially children, refuse to take proper nourishment; hence, substances tending to irritate the mucous membrane should be eliminated. Neutral food, such as lukewarm liquids or a soft diet, should be given. All spices should be excluded. Foods of extreme temperature and hard foods should be avoided. The bowels should be carefully regulated. In regard to local applications, no caustic, such as silver nitrate in any form or chromic acid, should be used. The best effect can be obtained by the application of a 2 to 3 per cent watery solution of dye products, such as gentiana violet, or Pyoktanin B, used for swabbing. Because of the constant presence of spirillae in the mouth, occasional swabbing with Fowler solution, glycerine and Vin ipecac in equal parts may be used. As a mouth wash, a mild neutral substance, such as bicarbonate of soda or aspirin, is advised. Very often sodium perborate and peroxide and other strong drugs cause further irritation. Some troches with anesthetic agents, such as anesthesin or orthoform, may help reduce the sensitivity of the mucous membrane. Many authorities believe that there is a vitamin deficiency present; therefore, an increased dose of vitamin B, cod liver oil, or orange juice (if tolerated), may aid toward a quicker recovery. In children, where local therapy may be very difficult, Flusser found an intracutaneous injection of milk and Aolan to be very effective in addition to general supportive treatment, which is very important in early childhood.

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LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY OFFICERS FOR 1939.

President: Dr. Pierre Viole.

Vice-President: Dr. Henry B. Lemere.

Secretary-Treasurer: Dr. John P. Lordan.

Committeeman: Dr. M. N. Beigelman.

Place: Los Angeles County Medical Association building, 1925 Wilshire boulevard, Los Angeles.

Time: 6 P.M., fourth Monday of each month from September to May, inclusive.

NYSTAGMUS: SOME OBSERVATIONS BASED ON AN ELECTRICAL METHOD FOR RECORDING EYE MOVEMENTS.*

DR. H. B. PERLMAN and DR. T. J. CASE, Chicago.

Recent studies by Mowrer, *et al.*, in 1936,[1] and by Fenn and Hursh[2] in 1937 extended the observations of Schott,[3] Meyer[4] and Jacobson[5] on the electrical phenomena associated with eye movements and indicated their value as a means of studying nystagmus. It has been shown by Mowrer and his co-workers that a constant potential difference exists between the front and back of the eye and that a movement of the eye is, hence, associated with a shifting of the resulting electrical field. The detection of these field changes by means of the string galvanometer as employed by Schott and his successors is associated with the difficulties inherent in this apparatus; notably low sensitivity and extreme fragility of the string. By means of the resistance capacity coupled vacuum tube amplifier and suitable recording apparatus, these potentials can be readily and simply recorded.[6] Silver discs are attached by means of electrode paste and collodion lateral to the outer canthi, and are connected by fine wires to the input of the amplifier. The amplifier has a conventional resistance capacity coupled circuit, the time constant of which should be made as large as practicable; *i.e.*, about one second. While a single-sided amplifier may be used for this purpose, if the patient is placed in a shielded room, the use of an amplifier with a balanced push-pull input avoids the necessity of shielding the patient. Most of the records here presented have been made with an ink-writing oscillograph having a natural frequency in the neighborhood of around 100 cycles per second. Such records have the advantage of being immediately visualized and are low in cost.

Methods of photographic recording are more expensive and more difficult to utilize but give a wave form which is slightly more accurate, depending upon the nature of the oscillograph

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employed. In general, such a setup as illustrated in Fig. 1 offers to the otologist an apparatus for examining and recording nystagmus which is rugged in construction and simple and inexpensive to operate.

The following figures show the electrical changes accompanying eye movements in a few selected clinical and experi-

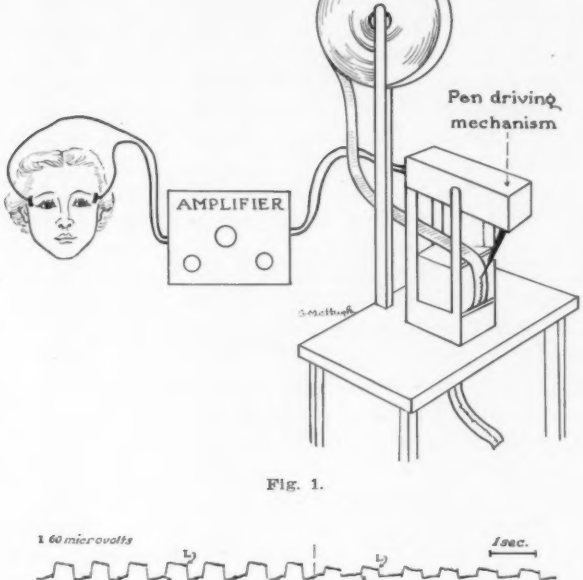

Fig. 1.

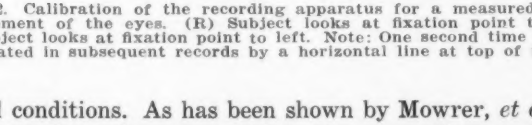

Fig. 2. Calibration of the recording apparatus for a measured lateral displacement of the eyes. (R) Subject looks at fixation point to right. (L) Subject looks at fixation point to left. Note: One second time interval is indicated in subsequent records by a horizontal line at top of figure.

mental conditions. As has been shown by Mowrer, *et al.*, the change in the voltage recorded in the plane of movement of the eye and at right angles to the optic axis is proportional to the product of the corneoretinal potential into the sine of the angle of displacement. Fig. 2 shows the change of voltage between horizontally placed electrodes, as described for an

angular deviation of about 12 degrees in the horizontal plane; *i.e.*, looking from right to left, and for an angular deviation of about 24 degrees. For recording vertical movements of the eye, the electrodes should be placed above and below the eye. Note that the displacement of the writing arm for an eye movement of 24 degrees is twice that for a movement of 12 degrees. An angular deviation as small as two degrees can be detected. Inasmuch as the corneoretinal potential varies from patient to patient,[1] the excursion of the recording needle per unit of angular deviation will vary in the same manner. The magnitude of the potentials recorded can be judged from the calibration indicating the displacement of the writing arm for 60 microvolts (see Fig. 2). The use of resistance capacity coupled amplifiers introduces a factor of decay in the amplifier which limits its applicability for recording very slow electrical changes. This decay of the amplifier is described by its time constant; this being the time taken for the recording needle to fall from a given deflection to one-half of that deflection, and is controlled by the ratio of the capacity of the interstage coupling of the amplifier to its associated resistance. In Fig. 2 the lateral deviation of the eye has been voluntarily maintained in the right and left position. That the record during this time period is not flat but slopes slightly is due to this decay factor. In general, the movement of the nystagmus and other eye movements which the otologist will wish to record are fast in comparison with this decay factor, which may then be neglected. But in those instances where slow eye movements are to be recorded, the decay period of the amplifier will become of importance, and under such circumstances the use of a direct coupled amplifier may be preferable. The use of a direct coupled amplifier obviates this decay factor, but such apparatus is a little more expensive to build and requires more care in its use.

Fig. 3A is a record of nystagmus produced in a normal young adult by irrigating the right ear with 10 cc. water at 23° C. The subject's eyes were open and he lay on his back. The number of oscillations per second is somewhat over two. The quick component lasts about one-twentieth of a second. The length of the slow component varies from three-tenths to five-tenths seconds. The angular displacement of the eye for each cycle is about four degrees (see Fig. 3C for the displacement per six degrees of eye movement in this indi-

vidual). Fig. 3B is a record of nystagmus in the same individual after using 10 cc. water at 9° C. as the stimulus and at about the same time interval (45 seconds) after the application of the stimulus as in Fig. 3A. The angular displacement of the eye at each cycle is about three degrees, the number of strokes per second is increased to almost three, the speed of the quick component is about one-twentieth second, and of the slow component between one-fifth and one-half second.

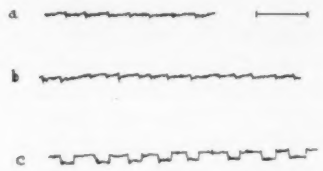

Fig. 3A. Record of nystagmus 45 seconds after irrigating right ear with 10 cc. water at 23° C.

Fig. 3B. Record of nystagmus 45 seconds after irrigating right ear with 10 cc. water at 9° C.

Fig. 3C. Calibration to show the displacement of the pen for 6° lateral eye movement in this individual.

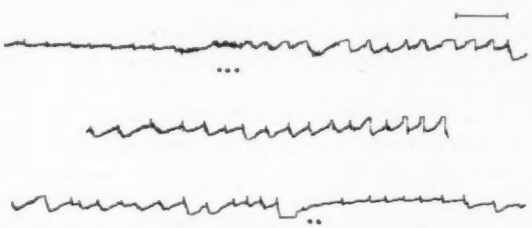

Fig. 3D. Continuous record of caloric nystagmus with the eyes open and with the eyes closed, beginning 45 seconds after douching the right ear with 10 cc. water at 23° C. Three dots (...) signals the command to close the eyes. Two dots (..) signals the command to open the eyes.

Fig. 3D is a record of nystagmus in the same individual, using a caloric stimulus of the same intensity as for Fig. 3A, but with the patient's eyes closed, thus avoiding fixation. The angular deviation for each stroke is increased markedly after closing the eyes to about two times that recorded with the eyes open, the duration of the quick component remains about one-twentieth second, and of the slow component between one-fourth and one-half second. The number of cycles of eye movements is decreased to less than two per second.

Note the sudden change in the character of the eye movements when fixation is present and when it is absent. Three dots (...) indicate where fixation is abolished by closing the eyes, and two dots (..) indicate where fixation returns when the eyes are opened.

In this record, as in many of the others here presented, a sharp spike begins the quick component. It occurs at the beginning of the quick component and is not to be confused with overthrow of the writing mechanism; cf. Schott (see Fig. 5). We believe, from recent studies, that this is due to action currents in the extrinsic muscles of the eye.

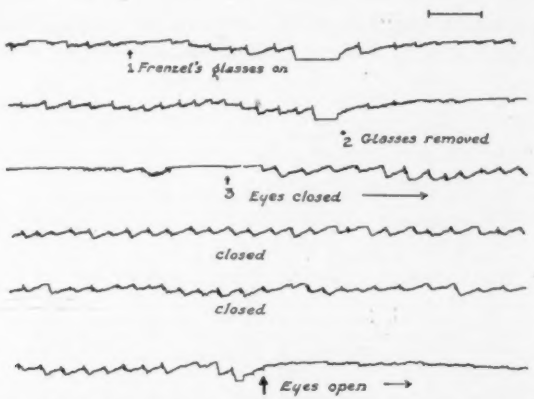

Fig. 3E. This is a continuous record beginning 45 seconds after irrigating right ear with 10 cc. water at 23° C., and runs for about 60 seconds, during which time the effect on nystagmus of applying Frenzel's glasses, closing and opening the eyes, was determined.

Fig. 3E is a record of nystagmus in the same individual indicating the effect of removing fixation by the use of Frenzel's glasses, and contrasting this with the effect on nystagmus of removing fixation by closure of the eyes. At (1) Frenzel's glasses are put on the subject 45 seconds after a caloric stimulus was applied to the right ear. The amplitude of eye movements is seen to increase; at (2) about 15 seconds after (1), the glasses were removed and abrupt marked diminution of the nystagmus is observed; at (3) the subject closed his eyes; note the quick return of a strong nystagmus, which is even greater than that recorded with Frenzel's glasses; at (4) the

eyes are opened again 30 seconds later and the nystagmus is seen to be practically gone. A factor to be considered in explanation of the difference in eye movement when fixation is removed by the use of Frenzel's glasses and when it is removed by closing the eyes, is the effect of light striking the retina on the nystagmus producing phenomena in the central nervous system.

Fig. 4A is a record of nystagmus due to stimulation of a labyrinthine fistula by pressure. The patient, a male, age 38 years, had a discharging left ear for five years, but gave a history of having frequent attacks of otorrhea in childhood. Examination revealed a large defect in the pars tensa, as well as in Schrapnell's membrane, with foul pus and cholesteatomatous debris. Raising the pressure in the external canal with a Politzer bag produced violent dizziness and nystagmus.

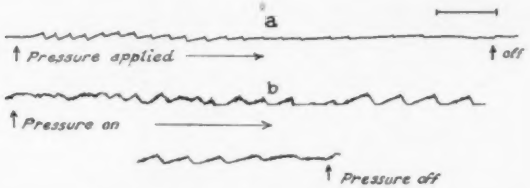

Fig. 4A and B. Record of vestibular reaction due to pressure on a labyrinth fistula. (A) With eyes open. (B) With eyes closed. (The fuzziness of the tracings is due to muscle action potentials, arising probably from contraction of the facial muscles.)

When the record was made the patient's eyes were open. Note the regularity of the nystagmus, and the gradual decrease in the amplitude and frequency of the eye movement. About nine seconds after the onset of the pressure the nystagmus has practically disappeared. The disappearance of nystagmus in the presence of a constant pressure against a fistula has long been known, being first described by Zytowitsch in 1913.[7] In a patient studied by Shambaugh,[8] nystagmus was found to disappear in 10 seconds with the fistula in a constant pressure field. He believed that this represented the time it took to collapse the membranous canal and cause deviation of the cupola to a position furthest away from its resting state. On release of pressure he noted a reversal of the nystagmus but with a duration equal to that seen on compression. "When pressure was applied for only a few seconds and then held,

that moment the nystagmus stopped." Nylen[9] has made similar observations. Further studies are planned with accurately controlled pressure to eliminate the objectionable factor of a gradual decrease in pressure taking place as a result of leakage of air at the external auditory meatus. The number of eye movements per second at the onset of nystagmus is almost four, the quick component lasts about one-twenty-fifth second, the slow component between one-fourth and one-sixth second.

Fig. 4B is a record of nystagmus in the same patient with a similar stimulus, but with the patient's eyes closed. Note the irregularity of the eye movements and their increased amplitude and decreased frequency. When rhythmic movements occur, their frequency is seen to be decreased to about two per second, and their amplitude to about twice that recorded with the eyes open.

Fig. 5. Record of pressure stimulation of a long-standing fistula in the left horizontal canal. At (X) the amplifier is blocked. Nystagmus continues during this time.

Fig. 5 is a record of nystagmus in a patient with a long-standing labyrinthine fistula, stimulated by pressure. This patient, a male, age 58 years, had a discharging ear for 38 years, and for many years had noted dizziness when cold air struck the ear or when he inserted cotton too far into the ear. Examination revealed a cholesteatomatous process which had destroyed the middle ear and mastoid structures to produce a cavity resembling that seen after a radical mastoid operation. The response to the fistula test was brisk. After cleaning the cavity, a dark streak could be visualized marking the site of the horizontal canal fistula. The beginning of the pressure stimulus applied with a Politzer bag is indicated in the record. Note the decrease in the speed and amplitude of the eye movements, similar to that seen in Fig. 4A. Six cycles of eye movements per second are seen at the onset, and less than two cycles per second at the end of the record, about nine seconds after the stimulus is started.

Fig. 6 is a record of violent nystagmus produced by pressure on a recent labyrinthine fistula. The patient, a male, age 30

years, had an earache about the middle of January. The earache recurred in a few days and discharge was noted in the canal. The ear drained for two weeks, when vertigo developed. The patient noted that he became momentarily dizzy when he blew his nose, sneezed, bent down or turned around quickly. He came in for examination about two months later, on April 6, when a perforation in Schrapnell's membrane with purulent discharge was found. Suddenly increasing pressure in the external auditory canal by means of a Politzer bag produced a violent nystagmus. Operation revealed a generalized softening of the mastoid by granulation tissue. The record shows the disorganization of eye movements that occur when

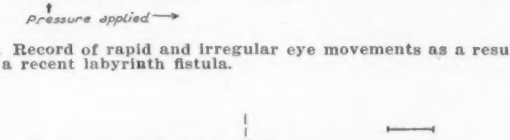

Fig. 6. Record of rapid and irregular eye movements as a result of pressure on a recent labyrinth fistula.

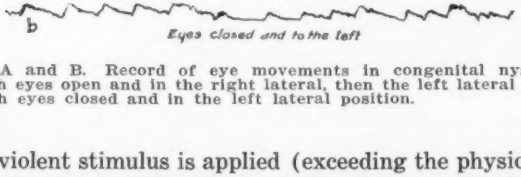

Fig. 7A and B. Record of eye movements in congenital nystagmus. (A) With eyes open and in the right lateral, then the left lateral position. (B) With eyes closed and in the left lateral position.

a very violent stimulus is applied (exceeding the physiological limit). Pressure on the fistula produced a very rapid, markedly irregular fibrillar movement of the eyes, which did not resemble the orderly rhythmic movements observed in the other records of nystagmus. This disorganization of eye movement following violent vestibular stimuli has been observed by Lorente do Nó[10] in the experimental animal.

Fig. 7A is a record of congenital nystagmus in a male, age 23 years. The peripheral vestibular and auditory mechanism appeared to be normal. His vision was moderately affected, although he had worked in a steel factory without any great difficulty. Examination revealed a horizontal nystagmus in the direction of gaze. The reversal of the wave forms when

the eyes are moved from the right lateral to the left lateral position is noteworthy.

Fig. 7B is a record of nystagmus in the above patient when the eyes were in the left lateral position and closed. The rhythmic movements of the eyes continue but their frequency has become less and their amplitude increased.

Fig. 8 is a record of nystagmus which was found to appear in a patient when he assumed a certain posture. This patient, a male, age 50 years, complained of attacks of dizziness for several years. He stated that he recently noted that he became dizzy only if he lay on his back. He could relieve this feeling by turning over to either side. Examination of the vestibular function by the caloric test disclosed nothing abnormal. When the patient was asked to assume the position in which dizzi-

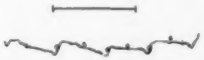

Fig. 8. Record of eye movement in position nystagmus. (Q) Represents the quick phase; (S) the slow phase.

Fig. 9A. Record of discrete eye displacements due to momentary pressure with a metal probe against site of fistula in the left horizontal canal.

ness occurred, a horizontal nystagmus to the left when the eyes were in the resting mid-position could be readily seen. The neurological examination was negative except for slight cerebral arteriosclerosis. On examination six months later this position nystagmus could not be elicited. "Q" indicates quick phase, and "S" slow phase of the nystagmus.

Fig. 9 is a record of the eye displacement resulting from discrete impulses applied to the perilymph at the site of a labyrinthine fistula of the patient, described in connection with Fig. 4. These stimuli were produced by lightly and quickly touching the fistula with a smooth metal probe. Pressure on the fistula located in the middle portion of the right horizontal canal produced a deviation of the eye to the left, and on release of pressure a return of the eye to the mid-position.

Fig. 9B is a record of discrete eye movements resulting from momentary pressure upon the fistula with a metallic probe. The slower deflection represents the movement of the eye similar to that seen in Fig. 9A; the first spike immediately preceding this results from equalization of the static electrical charges on the bodies of the patient and examiner at the moment of contact of the bare tip of the probe with the fistula (the shaft of the probe being insulated) and indicates, therefore, the instant when pressure upon the fistula begins. It is apparent that the latency of the response is short, probably less than one-hundredth second. This is of the same order of

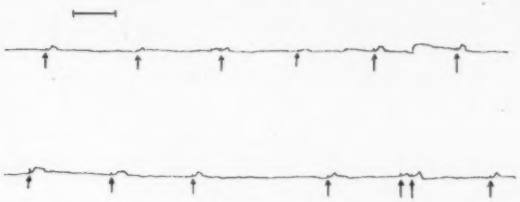

Fig. 9B. Record of discrete eye displacements produced as in Fig. 9A. Spike (see arrows) records the moment the probe touches the fistula.

Fig. 10. A continuous record of eye movement when subject rotates his head through about 100 degrees with eyes closed and with eyes open. Note that three or four large eye movements occur during each cycle of head rotation when the eyes are open. (R) Head is turned to the right. (L) Head is turned to the left.

magnitude as the latency determinations by Dohlman and Engvall[12] in the rabbit.

Fig. 10 is a record of eye movements which occur in a normal individual when he rotates his head, with respect to the trunk, about the vertical axis. In part (a) this rotation occurred with the eyes closed; in part (b) with the eyes open. When the eyes are open, fixation occurs three or four times when the subject turns his head about 100 degrees. With the eyes closed, such attempts at fixation appear to be indefinite.

Fig. 11A is a record of "searching movements" of the eyes in the mid-position, observed in a male, age 27 years, who

had great visual impairment as the result of congenital maldevelopment of the eyes, including bilateral congenital cataracts. This type of eye movement is usually seen in individuals born with a marked visual defect causing a failure in developments of the centres in the brain controlling fixation of the eye. The movement can best be described as an oscillation having equal or nearly equal time phases. The record resembles that of Miners' nystagmus[11] but is easily differentiated from the records of movement seen in vestibular nystagmus, where one phase may be 10 times as long as the other. A further contrast with vestibular nystagmus can be seen in Fig. 11B, taken with the eyes closed. These searching movements are seen to have practically disappeared (see Fig. 3E for the effect of closing the eyes on caloric nystagmus).

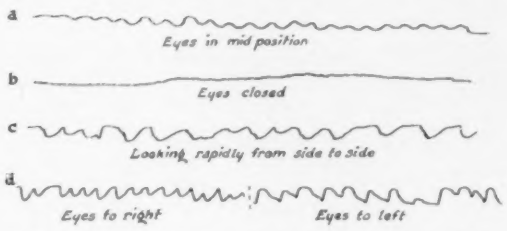

Fig. 11A, B, C and D. Record of "searching movements" of the eye under various conditions in congenital amblyopia.

Fig. 11C is a record of eye movements when the subject looks back and forth at two laterally placed objects, as in Fig. 2. Note the slow movement of the eyes to the lateral position, as indicated by the gradual slopes of the waves (compare with Fig. 2).

Fig. 11D is a record of spontaneous eye movement in the right and left lateral position. The equal time phase of the to-and-fro movement seen when the eyes are in the mid-position is changed to a slow and quick phase. The irregularity of the relationships between these phases and the amplitude of the eye movements is noteworthy.

SUMMARY.

A simple, rugged and economical method is described for recording nystagmus and similar eye movements. Records of

such eye movements are illustrated, and some are interpreted. Further investigations by this means of the factors producing these eye movements in the human, as well as in the experimental animal, should extend our knowledge in this relatively obscure field of neurophysiology.

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950 East 59th Street.

IN MEMORIAM

ANTOINE PHINEAS VOISLAWSKY, M.D.

1873-1939.

We sadly announce the death of our colleague in otolaryngology, Dr. Antoine Phineas Voislawsky, who died of a brain tumor at the Neurological Institute, New York, Feb. 22, 1939.

Dr. Voislawsky was born in New York 66 years ago, the son of Silas P. and Antonia Scharles Voislawsky, and practiced otolaryngology in his native city since 1898. He received his B.S. Degree from New York University in 1894, and graduated from the Medical School of Dartmouth College in 1897. At the time of his death he was Chief of Clinic and Attending Rhinologist and Laryngologist to St. Luke's Hospital, and Consultant to Flower and Fifth Avenue Hospitals, Northern Westchester Hospital, Staten Island Hospital, Hospital for Ruptured and Crippled Children, and the Orphans' Home and Asylum of the Protestant Episcopal Church. He was a member of the New York State and County Medical Associations; American Board of Ophthalmology, American Bronchoscopic Society and the American Board of Otolaryngology. He was a Fellow of the American Laryngological, Rhinological and Otological Society, the New York Academy of Medicine and the American College of Surgeons. He held various honorary fellowships, including the New York Academy of Dentistry and the Society of Alumni of St. Luke's Hospital and of the Men's Association of the Collegiate Church of St. Nicholas. He was President of the Alumni Association of New York University's Arts and Engineering Colleges, and a Director of the New York University Alumni Federation.

In addition to his successful otolaryngologic activities, he was interested in social science, the New York Zoological Society, the Seventh Regiment of the National Guard, and Constantine Commandery, Mecca Temple, Mystic Shrine.

His fraternities were Phi Gamma Delta and Phi Beta Kappa, and his clubs included the University, Bedford (N. Y.) Golf and Tennis, the Waccabuc Country Club at South Salem, N. Y., and the Phi Gamma Delta Club.

He is survived by his widow, the former Margaret Rutgers Van Rensselaer, of Belleville, N. J., whom he married on June 4, 1902; a son, Dr. Van Rensselaer Voislawsky; a daughter, Mrs. Van Rensselaer Wittmann. To the bereaved family we offer our sincere sympathy.

M. A. G.

NEW YORK ACADEMY OF MEDICINE.

SECTION ON OTOLARYNGOLOGY.

Meeting of Nov. 16, 1938.

SYMPOSIUM ON OTITIC MENINGITIS.

(a)—I. Diagnosis. Bacteriology of Suppurative Meningitis. Dr. A. A. Eggston.

(Published in January, 1939, issue of THE LARYNGOSCOPE.*)*

(a)—II. Diagnosis. Differential. Dr. J. G. Dwyer.

(Published in January, 1939, issue of THE LARYNGOSCOPE.*)*

(b)—Avenues of Infection. Dr. Marvin F. Jones.

(Published in January, 1939, issue of THE LARYNGOSCOPE.*)*

(c)—Operative Treatment of Meningitis. Dr. Wesley C. Bowers.

(Published in January, 1939, issue of THE LARYNGOSCOPE.*)*

(d)—Chemotherapy in Meningitis Secondary to Ear and Sinus Infections. Dr. Emanuel Appelbaum.

(Published in January, 1939, issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. JOSEPHINE B. NEAL: As I have been sitting here listening to these five extremely interesting papers, I have wondered what I could say that would add anything to the discussion; I am still wondering. I suppose the role to be played by one discussing the papers is not simply to praise, but in these papers there is very little with which I disagree, and I think the best I can do is to point out the things that I especially like.

I think Dr. Eggston can hardly be commended too highly for pointing out the impossibility of making a reliable diagnosis from a stained smear, and the necessity for a very careful cultural examination of spinal fluids. In so many laboratories we find that this is not done. Diagnoses are made from a stained smear. Quite recently I was called by a doctor who was treating unsuccessfully a case which he said was a meningococic meningitis. He finally sent us some specimens of the spinal fluid, and it was found to be an influenza bacillus. He did say the laboratory had some difficulty in growing the organism, but, from the smear, they thought it was a meningococcus, being misled by the coccoid forms the influenza bacillus so often takes. All these organisms may be highly pleomorphic in form on direct smear, and, as Dr. Eggston and Dr. Dwyer pointed out, often are irregular in staining. It does not seem to me that I have ever seen a meningococcus which took a Gram-positive stain, however, even directly in the smear.

There is nothing that I can add to Dr. Dwyer's discussion except to mention perhaps that occasionally the fluid in tuberculous meningitis may be distinctly

hazy and may show a preponderance of polymorphonuclear leukocytes. Fortunately, in these fluids the organisms are particularly numerous and the diagnosis is quickly made if one remembers to do a Ziehl-Nielsen as well as a Gram stain. At present we like growing the tubercle bacillus on a Bordet-Gengou culture medium better than inoculating the animals. Our bacteriologist finds the tubercle bacillus in smears of about 90 per cent of the cases, assuming that she is given a fair chance. Of course, if only 2 or 3 cc. are sent to the laboratory, one does not have much room in which to work around. I wish we could persuade the physicians to send us a larger amount of spinal fluid so that a more thorough examination could be made. It is, as both Dr. Eggston and Dr. Dwyer pointed out, of greatest importance that organisms be kept on culture media for a considerable period of time. Miss Gosling, our bacteriologist, does not discard them until at least two weeks have passed, and also as a routine makes a culture in a medium which will allow the growth of anerobia. Occasionally in that way we pick up a streptothrix — I believe nowadays we should say an actinomyces — which in the direct smear looks like a mixed infection.

Another point that Dr. Dwyer made which I certainly second most heartily is the removal of foci of infection when a serous meningitis develops. We see a great many of these cases, and we all have not infrequently seen instances where the prompt removal of the focus of infection while the spinal fluid, although cloudy, was free from organisms resulted in recovery without bacterial meningitis. We have been advocating this for years, incidentally.

Dr. Jones' very scholarly demonstration I can only admire and not discuss. I am very glad that he brought up the point of the explosive type of development of meningitis in which apparently the blood stream and spinal fluid are infected at approximately the same time, and, as he pointed out, that type of case is very highly fatal, even with the use of sulfanilamide and its derivatives. I certainly wish I knew as much about anatomy as Dr. Jones does. I think his presentation and slides were quite perfect, and I enjoyed them very much.

I am glad that Dr. Bowers emphasized that a mastoiditis may be present without tenderness, because so often we urge a mastoidectomy and have difficulty in getting the co-operation of the otolaryngologist. The point was emphasized both by Dr. Bowers and Dr. Appelbaum, and also the fact that in addition to the possibility of a mastoiditis, or even the occurrence of a mastoiditis, there may also be involvement of some of the accessory sinuses, and hence the necessity of operating on them. I feel very strongly that one reason we have not been getting better results in cases of pneumococcic meningitis with neoprontosil is that it has been impossible to demonstrate adequately a focus of infection. I can only hope that some new technique or X-ray examination will be developed to make it possible for these areas of infection to be diagnosed more quickly. It seems to me that it is very desirable to operate on foci of infection even after the organism has invaded the spinal fluid and we have a real bacterial meningitis. As Dr. Appelbaum said, a number of our cases have gotten well without the focus of infection being located and removed. I always feel rather anxious about the future of those patients. To be sure, as Dr. Appelbaum quoted from Marshall, Emerson and Cutting, experimentally, there may be penetration of the bone by sulfanilamide products. I must confess that I do not trust too much in neoprontosil's adequately clearing up a really severe focus of infection. It may quiet down and the patient may recover from his meningitis, but I know of one instance where, after quite a period of time, a second attack of streptococcic meningitis developed in a boy who had been in Willard Parker Hospital. Fortunately he recovered from this attack as well. Previously a recovery from even one attack would have been a cause for celebration.

I am certainly very glad to have lived to see this day when these forms of meningitis are not as deadly as they were. It seems to me that the results in streptococcic meningitis are really wonderfully good, considering all the pos-

sibilities that are involved. And the results in pneumococcic meningitis — well, how much better is seven out of 42 than none out of 200 or 300! I believe that when one divides by zero as a denominator one gets quite a large quotient.

Dr. Philip D. Kerrison: It should be sufficiently obvious, I think, that one who has been so long out of practice as I is not qualified to discuss these exceedingly interesting papers. I can only say that in a recent attempt to inform myself on the general theory and method of the prontylin treatment of otitic meningitis, the effect on me personally was an enthusiastic sense of confidence in the future potentialities of otology. Aside from the almost fantastic results as shown in the reversal of recent mortality and recovery statistics in otitic leptomeningitis, I think that as a by-product the stimulation and increased confidence of present day workers in otology should be second only in value to the spectacular advances recently obtained. In a day of world-wide confusion, medical science, and with it otology, goes on and will not stop.

NEW YORK ACADEMY OF MEDICINE.

SECTION OF OTOLARYNGOLOGY.

Meeting of Dec. 21, 1938.

a. Deep Infections of the Neck.

1. Anatomy. By Dr. John M. Loré.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

2. Diagnosis and Treatment. By Dr. Henry B. Orton.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

3. Discussion. By Dr. Henry S. Dunning.

b. Facial Paralysis.

1. Nerve Graft Operation. By Dr. Thomas G. Tickle.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

2. Plastic Reconstruction. By Dr. Clarence R. Straatsma (by invitation).

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. HENRY S. DUNNING: It has given me great pleasure to listen to these two very interesting papers. It is a subject that I am most interested in, and I wish to congratulate the two essayists upon their splendid presentation.

In talking about mouth infections from the standpoint of an oral man, I would like to ask the two men that preceded me what they think would be the per cent of mouth infections and how many nasal pharyngeal infections travel into the floor of the mouth and tongue regions. Working in the mouth and throat, I find a great many infections of the floor of the mouth and tongue and tonsillar regions are due to some kind of dental operation, most often from post-extraction wounds. We find at the oral surgery clinic, Columbia Medical Center, that we have some very severe sequelae develop and at times we lose a patient from the lack of early treatment, and sometimes we lose them in spite of very good early treatment. It seems to me that when a severe oral infection starts we are, in many cases, dealing with dynamite. I feel that we should get these cases into the hospital at once, and that they should have a very careful workup, and that adequate drainage should be established as soon as possible. We should not attempt to treat these cases in a clinic. We have had a number of such cases treated for three or four days in a clinic, involving the tongue and floor of the mouth, that developed later into a real Ludwig's angina following a tooth extraction. I think these cases should be opened up very radically by a large lateral incision and careful neck dissection, exposing thoroughly the submandibular triangle. We should open up very thoroughly whether we get pus or not, thus relieving tissue tension and hoping that the pus will drain into the incision in a day or two. Even if we do not strike oil at the time of operation, I believe that radical surgery and careful treatment will save these cases at times, if we have any luck at all, but occasionally nothing seems to help.

I do not know whether the medical men realize the importance of the pericoronal flap over the third molar in the extraction of wisdom teeth. It is a very short distance from the flap of gum around the wisdom tooth to the capsule of the tonsil. Many peritonsillar abscesses are caused by the infiltration of pus from a dirty gum flap in the tonsillar region. The areolar tissue becomes infected very easily when the gum is traumatized in the extraction of a tooth. It is very hard to keep these wounds clean in the lower jaw, and they drain into the peritonsillar region quite frequently. The average dental surgeon often is called upon to treat this condition and at times does not recognize the situation. I believe that these cases should be handled by a nose and throat surgeon. Sometimes by inserting a curved hemostat into the third molar wound, and turning it inwards over the internal oblique line along the inner surface of the ramus, the peritonsillar abscess can be drained very nicely without making the regular incision generally used in these cases. Many times just sticking a forceps in the wound as described above and opening the jaws of the forceps will give sufficient drainage to abort a peritonsillar abscess. These infections are quite common in oral surgery practice.

We have been using in conjunction with these mouth infections zinc peroxide, advocated by Dr. Frank Meleney. I often smear it around in the mouth before operation on a particularly dirty mouth case, and use it quite regularly as a postoperative measure. The zinc peroxide makes a very good mouth wash, 25 per cent, and it is very efficacious when placed in a dirty wound. It slowly liberates oxygen, and cleans up many of these cases very nicely.

We feel that the jaw bones are infected more often than any other bones of the body, directly from a dirty cavity—the mouth—and a great many of these neck and floor-of-the-mouth infections are very prevalent in the practice of the oral surgeon. There are two main routes which the infection travels to the teeth—one, the peridental route around the tooth to the gums, under the periosteum and soft tissues, and the other, the intradental route, through the dead nerve canal to the apex of the tooth and cancellous bone, thence under the periosteum and soft tissues.

BOOK REVIEWS.

Surgery of the Ear. Samuel J. Kopetzky, M.D., F.A.C.S., Editor; Professor of Otology, New York Polyclinic Medical School and Hospital, New York. Four hundred sixty-eight pages, 292 illustrations and four color plates. New York: Thomas Nelson and Sons.

In the past decade there have been so many advances in the surgical treatment of otologic disease that a new book, compiled with a view to bringing Otologic Surgery up to date, is not untimely. It is edited by Dr. Samuel J. Kopetzky, which, in itself, is an index of its scientific and literary value. There are 18 special chapters, each contributed by an outstanding authority, and each monograph is presented in masterful form.

The volume is bound in loose-leaf manner, permitting the addition of new accumulated material. Perhaps it may be well to describe this reference volume as a series of correlated monographs, each minutely describing surgical diagnosis and operative technique, and with excellent illustrations, the entire series forming a splendid symposium of otologic surgery.

It is perhaps the most outstanding reference volume on surgical technique in otology that has as yet appeared in American literature, and to Dr. Kopetzky and his able and well selected collaborators we extend our congratulations on this valuable contribution.

It is hardly to be regarded as a textbook but rather as a reference volume for the master otologist. We know of no similar publication in English that presents modern otologic surgery in so complete and so-to-date form.

M. A. G.

Diseases of the Nose, Throat and Ear. W. Wallace Morrison, M.D., Clinical Professor and Chief of Clinic, Department of Otolaryngology, New York Polyclinic Medical School and Hospital. Three hundred thirty-four illustrations; 675 pages with index. Philadelphia: W. B. Saunders Co., 1938. Price $5.50 net. Cloth.

Dr. Morrison adds one more to several new books devoted to the field of Otolaryngology. It is essentially a textbook, but omits considerable detailed anatomical and physiological material which is frequently included in more extensive works. It nevertheless fulfills in many ways the essential requirements of a comprehensive treatise for daily use by the clinician. The subjects of history taking and physical examination have received particularly satisfactory attention and treatment is adequately covered within the limits of a book dealing with otolaryngology as a whole. Descriptions of operative procedures are intentionally limited with a view to providing information rather more as to what the patient requires than as to how any specific operation should be performed.

The 650 pages of text are illustrated by 350 line drawings, all from the author's own pen. The labor of such personal illustration is patently colossal and deserves greater success than has, in this reviewer's opinion, been achieved. This fact results primarily from an attempt in a single drawing to show too many individual items with resultant multiplicity of designation which is confusing. Such a type of illustration involves extensive legends, the searching of which provokes fatigue and at times even discouragement.

Admirable innovations are special indices of symptoms and a most comprehensive and useful formulary for medical treatment of many common

diseases. The book is avowedly a compilation of the author's lectures to post-graduate students with the prefaced declaration that the book is nevertheless intended primarily for undergraduate medical students and general practitioners. With this declaration this reviewer cannot agree. The book on diseases of the ear, nose and throat entirely suited for these two classes of readers has yet to be written and cannot be modeled along the classical text-book lines which characterize Dr. Morrison's book. That the latter will prove of great value to post-graduate students and specialists there can be no question.

L. R.

Practical Otology, Rhinology and Laryngology. By Adam Edward Schlanser, M.D., Colonel, Medical Corps, U. S. Army; Chief of the Eye, Ear, Nose and Throat Service, Letterman General Hospital, San Francisco; former Director of the Department of Ophthalmology and Otorhinolaryngology, Army Medical School; Chief of the Eye, Ear, Nose and Throat Section, Walter Reed General Hospital, Washington; Member of the Association of Military Surgeons of the United States; Fellow, American College of Surgeons and of the American Academy of Ophthalmology and Oto-Laryngology; Chevalier of the Legion of Honor. Illustrated with 91 engravings, 315 pages with Index. Philadelphia: Lea & Febiger, Washington Square, 1938. Price $4.50.

It is difficult to justly appraise the qualities of "practical" treatises on medical subjects. Invariably, in the author's attempt to be brief, some of the very important features of the subject are omitted; and often enough, his enthusiasm for certain phases of the contained material carries him beyond the limits of the "practical."

The author states in his preface that his work is largely the result of an effort to acquaint medical officers in the military service with the fundamentals of otology, rhinology and laryngology to the end that they may, if called upon, be reasonably competent in the management of diseases in this field.

Some of the chapters are too detailed for such a purpose. For example, nasal deformities and their correction are given much more space than is accorded them in most of the standard texts. It is not reasonable to suppose that problems of this nature should fall in the province of the casual medical officer who should happen to chance upon them. But, on the other hand, such an officer may be expected to remove an insect or a collection of impacted cerumen from the ear of one of the men in his company; and yet there is nowhere to be found a word on foreign bodies of the ear.

It is not to be inferred, however, that the volume is not a creditable contribution. In general, the material is well planned, intelligently and lucidly exposed, and, what is more important, it is to the point. The most interesting chapter is that on deafness and the simulated deafness which in the military service must assume considerable importance.

E. J. L.

The Acoustic Method for the Training of the Deaf and Hard-of-Hearing Child. By Max A. Goldstein, M.D., LL.D., Director, Central Institute for the Deaf; Professor of Otology, St. Louis University, 1900-1912; President, American Academy of Ophthalmology and Oto-Laryngology, 1902; President, American Otological Society, Inc., 1928; President, American Laryngological, Rhinological and Otological Society,

1931; President, National Forum on Deafness and Speech Pathology, 1917-1939; President, American Speech Correction Association, 1936-1938; Editor. THE LARYNGOSCOPE. With 246 pages and numerous illustrations. St. Louis: THE LARYNGOSCOPE PRESS, 4574 West Papin street. Price $3,00 postpaid.

Dr. Goldstein, in his varied experience as a practicing otologist and as director of the Central Institute for the Deaf, has long been one of the principal advocates of the utilization of residual hearing in the education of the deaf child. This volume presents the philosophy of that idea and in considerable detail explains the methods used in this educational approach.

If, as he states, "over 30 per cent of reported congenitally deaf cases exhibit a residuum of hearing amenable to re-education and stimulation," this residual hearing should be used to a maximum. He states, "The Acoustic Method, or that phase of it that applies to the stimulation of hearing by voice or musical instruments, has heretofore been applied in greater part only to partially deaf children whose hearing capacity for spoken language is still adequate." He advocates that this method be extended, "not only to the partially deaf child . . . but also to the child who shows residual hearing of a very limited degree, insufficient to comprehend language and without the natural resources for language and hearing progress."

The volume is prepared primarily as a text for educators. In Chapter V, teaching methods are presented by Mrs. Smalley, Miss Fogle, Mrs. McCall and Mrs. Silverman, teachers at the Central Institute for the Deaf, who have used this method for a number of years. The manner of presentation is clear and concise.

Throughout the text, the author presents the facts he has secured from his otological experience which apply directly to the education of the deaf child. The two chapters on hearing tests are more complete than are presented in many modern otological texts. A chapter on anatomy and physiology, while necessarily brief, presents these subjects in an understanding manner for those not expecting to become trained anatomists but who do need a rather complete knowledge of the structure and function of the hearing and voice mechanisms.

The volume is a worthy successor to the author's "Problems of the Deaf."

C. C. B.